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THE UNIVERSITY OF ALBERTA

THE EFFECTS OF LOCAL FOREARM COOLING ON CUTANEOUS AND SKELETAL MUSCLE BLOOD FLOW

by

PHILLIP JOHN HANDCOCK

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF MASTER OF SCIENCE

PHYSICAL EDUCATION AND SPORT STUDIES

EDMONTON, ALBERTA

SPRING 1986:

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ISBN 0-315-30223-2

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Date April 15, 1986

Dedication

To my family Moira, Jack and Gerry, for their continued support and encouragement. Their contributions have been immeasurable, but without them, this thesis would never have been attempted or completed.

Abstract

The purpose of this study was to examine the effects of two different temperatures of therapeutic cold, on skeletal muscle blood flow, and cutaneous circulation of the forearm. Sixteen male volunteers, aged 20 to 33 yrs. (mean age = 25.55 ±3.35 yrs.), were tested on two occasions with a different water immersion temperature used for each test. The therapeutic temperatures used were 5 °C and 10 °C, and were assigned randomly to each subject. The subject's right arm was used for all experimental procedures. Proximal and distal blood flow were estimated simultaneously, using mercury in silastic strain gauge venous occlusion plethysmography. It has been established elsewhere, that proximal and distal blood flow measurements, are representative of muscle and skin circulation respectively. Plethsymographic recordings were made every minute for 10 min. prior to treatment (control), 30 min. during immersion (experimental), and for the 30 min. following immersion (post-experimental).

A series of two way analysis of variance tests with repeated measures over both the time and temperature factors were utilised to analyse the data. The results indicated that both treatment temperatures, significantly reduced the proximal and distal blood flow throughout the 60 min. treatment period. A significant temperature effect was demonstrated for the proximal blood flow during the experimental period. During this period, the 10 °C treatment reduced proximal blood flow more than the 5 °C treatment. Distal blood flow was not, however, influenced by the treatment temperature. The 5 °C treated forearm exhibited a reactive hyperemia at the proximal and distal sites, but this reaction was not observed, proximally or distally, in the 10 °C treated forearm.

Previous studies have indicated that the cutaneous blood flow and the skeletal muscle blood flow of limbs, respond independently to various stimuli. Results from the present study, suggest that during the 10 °C treatment, the skin and muscle blood flows both appear to decrease. However for the 5 °C treated forearm, although the cutaneous blood flow is reduced, the vasoconstriction of the skeletal muscle blood flow is reduced, and tends to counteract cutaneous changes.

Information is provided in the present study, that suggests that a 10 °C cold treatment produces a more effective reduction in forearm blood flow, than the 5 °C treatment and does not elicit a reactive hyperemia.

Acknowledgements

The author gratefully acknowledges the individual and collective contributions of his committee.

To Dr. Steve Hunka, for his valued contributions with the statistical analysis and interpretation of the findings.

To Dr. Wally Cottle, for his many hours spent in careful explanation and construction of the apparatus. Without his specialised input, this project could not have been conducted in this manner.

The author would also like to express his sincere thanks to supervising Chairman, Dr. Steve Mendryk, whose patient counsel and advice helped to bring the original idea to a research conclusion.

Special thanks also to Dr. Dwight Harley of the Division of Educational Research Services, for his painstaking and enlightening work with the statistical analysis and graphics.

Thanks go to all of my fellow graduate students, and friends at the University of Alberta, who were a constant source of information, ideas and entertainment.

And finally, to my fiancee Deb, who provided me with a loving supportive environment for the completion of this research.

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I. INTRODUCTION

A. The Research Problem

Cold therapy has become the preferred initial treatment for acute soft tissue sporting injuries, over the last 20 years (Chu & Lutt, 1969, Barnes, 1979, Kramer & Mendryk, 1979). Such therapy is also popular in the latter stages of rehabilitation (Grant, 1964, Lee & Warren, 1978).

The trauma from athletic injuries causes damage to local vascular structures, allowing direct hemorrhaging into the interstitial spaces. The inflammatory reaction to such injuries, tends to compound the primary damage suffered, by increasing blood flow to the injury site, and promoting edema formation, through increased capillary permeability (Knight, 1976, Lee & Warren, 1978). Much of the inflammatory response to soft tissue injury is considered to be an overreaction (Wise, 1973), creating excessive edema and hematoma formation. An earlier return to activity can be ensured, if the inflammatory reaction can be limited, and the subsequent hematoma reabsorption can be facilitated.

It is claimed that local cold application will mediate a local vasoconstriction, limiting hemorrhage and hematoma formation. It is also suggested that cold will reduce the metabolic needs of the injured tissues, reduce muscular spasm, and reduce pain associated with the injury (Edwards, 1971, Kalenak et al, 1975, Knight, 1976, Barnes, 1979, Kramer & Mendryk, 1979, Hocutt, 1981).

Logically, cold treatment should minimise the local consequences of injury, and a smaller hematoma should result. From a rehabilitation perspective, healing will be further encouraged, if the hematoma reabsorption can be facilitated. Traditionally, heating modalities have been favoured to supposedly increase circulation to the injury site, improving nutrient delivery, and hastening the reabsorption of edematous fluid and tissue debris. Cold treatment in conjunction with local exercise, has been advocated as potentially useful in these rehabilitative stages (Edwards, 1971, Wise, 1973). Cold through various mechanisms, has been shown to significantly reduce pain sensation (Benson & Copp, 1974). The partial anesthesia

afforded by cold treatment permits gentle exercise of the injured parts. This exercise allows an improvement in injury mobility, an increase in blood flow to the exercising parts, and greatly assists lymphatic drainage. It is also claimed that circulation can be further increased, by a local reflex vasodilation to cold (Chu & Lutt, 1969, Edwards, 1971, Behnke, 1974). This effect is supposedly achieved, by manipulating the duration of the cold application.

The theoretical basis for primary care cold therapy, and late rehabilitative cold therapy, is provided has been based on the findings of Lewis' (1930) classical study. Lewis observed that an initial vasoconstriction of 9 to 16 minutes duration, occurred in the cold immersed finger. This initial vasoconstriction was followed by 4 to 5 minutes of vasodilation, until replaced by a further vasoconstriction. This cycling of local blood flow was termed by Lewis, " the hunting response". For acute soft tissue injury treatments, cold application attempts to avoid or minimise this hunting response, by limiting the duration of cooling. Rehabilitative cold therapy, however, attempts to take advantage of the hunting response, by prolonging cold treatment until vasodilation occurs (Edwards, 1971, Wise, 1973). Knight (1980) claims that Lewis' (1930) findings are often misinterpreted by cold therapy exponents. Three points about Lewis' study should be considered when generalising from his results. Firstly, the study was conducted on a highly vascular fingertip, rich with arterio-venous anastamoses. Secondly, the fingertip has an atypical composition compared with most body parts, in that it is largely skin and bone with very little underlying skeletal muscle. Finally, Lewis measured skin temperature and not deep blood flow. In light of these considerations, it would appear that many of the theories on cold therapy may be the result of overgeneralisations.

Experimental work on superficial heating (Roddie et al, 1956), and superficial cooling (Clarke et al, 1958), have demonstrated that cutaneous and skeletal muscle blood flow respond independently to various stimuli. Clarke, Hellon, and Lind (1958), in a forearm cooling experiment, found that limb blood flow actually increased gradually with time, and that this increase was predominantly in the forearm muscle. These findings cast doubt on the proposed effects of cold on deep blood flow.

As the majority of disabling soft tissue athletic injuries are to the deeper structures of the body, it is advantageous for therapists to be aware of the effects that cold will have on blood flow to the deep as well as to the superficial structures.

B. The Purpose

The purpose of the present study, was to investigate the response of forearm circulation to varying water bath temperatures. Venous occlusion plethysmography with mercury in silastic strain gauges, was used to measure limb blood flow. The measurement of volume changes in limbs, has long been used for the estimation of peripheral blood flow (Hewlett & van Zwaluwenberg, 1909). Whitney (1953) adapted the mercury in rubber strain gauge for plethysmographic estimation of limb blood flow. This technique has been validated by simultaneous water plethysmography (Dahn and Hallböök, 1970), and by extracorporeal perfusion (Englund et al, 1972). This method is based on the assumption that the length of a limb is fixed, therefore any increase in limb volume will be manifested in an increased transverse cross sectional area, at any point along the limb length. The percentage change in the sectional area will equal twice the percentage change of the circumference of that section, as measured by changes in the mercury strain gauge resistance (Whitney, 1953).

The forearm is an easily accessible and suitable limb for study, containing approximately 60% skeletal muscle (Hellon, 1963). Clarke, Hellon, and Lind (1958), noted that the distal portion of the forearm is predominantly bone and cutaneous tissue, whereas the proximal section is largely muscular tissue. These workers have demonstrated that distal and proximal strain gauges, reflect skin and skeletal muscle blood flow respectively. This technique of blood flow estimation was adopted for the present study, in an attempt to partition cutaneous and muscular circulation in the forearm. Proximal and distal strain gauge plethysmography of the forearm was particularly suitable for the present study, as it was non-invasive, and permitted comparisons of blood flow over an extended period of time.

Thirty (30) minute immersions in 5 °C and 10 °C water baths were conducted, with blood flow being monitored during this time and for another 30 minutes following the

immersion. In this way blood flow responses to cold, and the after effects of cooling on the circulation were monitored. Cold tap water was found to be approximately 10 °C under most conditions, and the water tank temperature could not be maintained below 5 °C.

C. Research Hypotheses

Four null hypotheses were tested at the 0.05 level of significance as follows:

1. That there will be no significant difference between the 5 °C and 10 °C treatment effects, on proximal blood flow.
2. That there will be no significant difference between the 5 °C and 10 °C treatment effects, on distal blood flow.
3. That there will be no significant difference in proximal blood flow, from pretest proximal blood flow, over time.
4. That there will be no significant difference in distal blood flow, from pre-test distal blood flow, over time.

These hypotheses will be examined in the context of the experimental period, the post-experimental period, and the experimental and post-experimental periods together as a single treatment period.

D. Limitations of the Study

The limitations of the present study include:

1. The accuracy of skeletal muscle and cutaneous blood flow measurement by proximal and distal strain gauge plethysmography respectively.
2. The assumption that the expansion of the forearm with venous occlusion was radially isotropic.
3. That the high compliance of the mercury in silastic strain gauges, resulted in no tissue deformation.
4. That a linear relationship between change in gauge length and change in gauge resistance existed.

5. That temperature changes did not significantly alter the strain gauge sensitivity and subsequent blood flow calculations.
6. That forearm girths did not significantly alter during the experiment.

E. Delimitations of the Study

The present study was delimited by the following factors:

1. The use of normal, male, university aged volunteers, with no cardiovascular anomalies.
2. The use of subjects living in a cold ambient environment.
3. The measurement of blood flow with mercury in silastic strain gauge plethysmography.
4. The two water bath temperatures used in the study, of 5 °C and 10 °C.
5. The recording of blood flow for 10 min. prior to cold immersion, 30 min. during cold immersion, and for 30 min. following the immersion.

F. Definition of Terms

Plethysmography:

The measurement of volume changes. For the present study, forearm volume changes were monitored during venous occlusion, for the estimation of forearm blood flow.

Vasoconstriction:

Refers to a decrease in local blood flow, caused by a decrease in the diameter of the vasculature.

Vasodilation: Refers to an increase in local blood flow, due to an increase in the diameter of the local vasculature. Previous studies make no reference to resting blood flow levels, so that vasoconstriction and vasodilation are not relative terms, and merely indicate a change in blood flow.

II. REVIEW OF THE LITERATURE

A. The Skin

Circulation

The cutaneous circulation is highly variable according to the region of the body, the age of the person, the existence of adjacent structures, and the environmental stresses (Sparks, 1978).

Skin consists of two main layers, an epidermis, which is the waterproof layer of cells in varying stages of keratinisation, and an inner layer known as the dermis or corium. The dermis is the connective tissue layer supporting the epidermis, and contains blood vessels, lymphatics, sensory nerves and receptors, sweat glands and hair follicles (Marples 1965, Roddie 1983). Deep to the dermis is the subcutaneous layer of connective tissue, fat cells and nerves.

Arteries serving the skin enter the subcutaneous layer, where they branch and anastamose freely to form a horizontal subcutaneous arterial plexus. Small branches of the subcutaneous arteries give rise to a capillary network between the fat cells, while others penetrate the dermis to form a dermal arterial plexus. Capillary networks again form from branches, to surround sweat glands, fat cells, and hair follicles. Arterioles ascend from the dermal arterial plexus to form a further horizontal plexus of metarterioles in the sub papillary region. Metarterioles or capillaries ascend from this to form hairpin-like loops into the papillae. The distal capillary descends to join a subpapillary venous plexus. Venules descend from this level into the small veins of the dermal venous plexus, paralleling the arterial vasculature back to the deep veins. Many interconnections or short circuits may occur within this system, but those described above are the principle plexuses (Sparks, 1978, Millington et al, 1983).

The smooth muscle coats of the arterioles gradually become thinner as they ascend, until the capillaries and venules are left with no smooth muscle coat at all. The capillaries and

venules do however contain endothelial cells which could have contractile properties (Sparks, 1978).

Arterio-venous anastamoses are found in certain areas of the body only. These are thoroughfare channels, with several layers of smooth muscle coat, able to shunt large volumes of blood . According to Roddie (1983), arterio-venous anastamoses have not been identified in the skin of the forearm.

Precapillary sphincters have been isolated in the cutaneous circulation, and are capable of controlling blood flow through the capillary beds (Millington et al, 1983). The other cutaneous vascularity of note, is a lymphatic plexus originating in the papillary region, and running parallel to the venous system back towards the body center.

The cutaneous circulation subserves two main functions, firstly the nutrition of the skin tissue, and secondly the conduction of heat from the internal structures of the body to the skin (Greenfield et al, 1963). Skin blood flow, in most situations, far exceeds the metabolic needs of the skin (Millington et al, 1983). During severe heat stress, the skin blood flow can account for up to 50% of cardiac output, or it can be reduced to virtually zero on cooling (Roddie, 1983). A large function of the skin circulation is heat exchange. Roddie (1983), claims that the most efficient heat exchange occurs at the terminal capillary loops which lie closest to the skin, and boast the greatest surface area. Other heat exchange mechanisms, include an extensive subcutaneous venous plexus, capable of holding large quantities of blood to heat the skin surface, and the arteriovenous anatamoses. These structures are capable of flooding the subcutaneous venous plexus directly with arterial blood, speeding the exchange of heat (Guyton, 1976).

Control of Circulation

The control of blood flow to the various vascular beds is dependent on the reactivity of the smooth muscle cells of the small arteries, arterioles, precapillary sphincters, arterio-venous anastamoses, venules, and veins. The smooth muscle cells of the small resistance vessels and veins, often display spontaneous contractile activity, are stretch sensitive

and respond directly to humoral and physical stimulation (Marshall, 1980). Smooth muscle sensitivity to blood borne or neurotransmitter substances will depend on the distribution of alpha and beta receptor sites. The so called beta receptors, are generally associated with vasodilation and have a high affinity for adrenaline. However, in general alpha receptors are excited mainly by noradrenaline and mediate a vasoconstriction (Marshall, 1980, Little, 1981).

The arterioles, arterio-venous anastamoses, venules, and particularly the veins of the cutaneous vascular bed, are innervated by sympathetic adrenergic nerve fibers (Sparks, 1978, Rowell, 1983). It appears that beta receptors in the cutaneous vessels are rare, and probably do not exist at all (Rowell, 1983). The sympathetic adrenergic fibers are seldom quiescent, according to Vander, Sherman, and Luciano (1980), and consequently always maintain some degree of vasoconstrictor tone in the skin vessels. This appears to be true for many of the cutaneous areas, most notably, those areas with abundant arterio-venous anastamoses. Blood flow is reduced in these areas, which tend to be the distal parts of the anatomy (acral), by increasing vasoconstrictor tone. An increase in blood flow is brought about by a reduction in vasoconstrictor tone (Roddie 1963).

It is estimated by Sparks (1978), that in thermally comfortable subjects, approximately one half of the total skin blood flow is to the hands, head, and feet (Sparks, 1978). These acral areas are rich in arterio-venous anastamoses, and therefore are able to alter blood flow rapidly in response to varying sympathetic adrenergic discharge (Sparks, 1978).

As described previously, the cutaneous vascular bed of the forearm has few arterio-venous anastamoses, and therefore has a relatively low resting blood flow. The forearm skin, however, has an active vasodilator mechanism mediated through sympathetic fibers (Roddie, 1983). These fibers release acetylcholine, which as well as having a direct dilator effect on the vessels (Whelan & Skinner, 1963), provokes sweat gland activity (Greenfield, 1963). The sweat formed contains a bradykinin forming enzyme, which acts on the protein in subcutaneous tissue spaces, to form bradykinin (Roddie, 1983). Bradykinin is a potent vasodilator, which diffuses into the blood vessels in the immediate area (Downey, 1964). While vasoconstrictor tone may be the key to blood flow control in many parts of the

skin, in the forearm the vasodilator mechanism is the main regulator of cutaneous circulation (Fox & Edholm, 1963). In fact in comfortably warm subjects, the vasoconstrictor tone in the forearm cutaneous vessels, may be fully released (Roddie, 1983).

Factors Influencing Circulation

Skin blood flow can range from 0 - 75 ml./100 ml. of tissue/min. (Sparks, 1978), depending upon the environmental conditions and the homeostatic responses within the body.

Exercise

The effect of exercise on the blood flow in uninvolved vascular beds, has been closely studied (Brengelmann et al, 1977). In response to lower limb exercise there is a transient initial vasoconstriction, followed by a longer vasodilation. The initial rise in arterial pressure during exercise, is thought to be the result of the vasoconstriction (a smooth muscle response to stretch), and the response to increased internal body temperature, for the eventual vasodilation (Roddie, 1983).

Changes in pressure

The cardiopulmonary system contains various baroreceptors responsive to pressure and postural changes. These receptors regulate blood flow to maintain suitable perfusion pressures. The influence of these receptors on cutaneous blood flow has not been satisfactorily defined (Mancia & Mark, 1983), although it is not thought to be a major effect.

Emotional stress

Severe emotional stress causes a vasodilation in skeletal muscle, but it's effect on the cutaneous circulation is uncertain (Roddie, 1983). In the hands and feet (acral regions), an initial vasoconstriction occurs in response to an alerting stimulus. However in nonacral skin segments, there does not appear to be any reaction. It is possible that sweating in response to stress, triggers a vasodilation which balances any vasoconstriction that does occur (Roddie, 1983)

Temperature

As described above, one of the major functions of the cutaneous circulation is that of heat exchange. The dramatic effects that changes in local temperature have on skin blood flow, are therefore not unexpected.

The core temperature of the body should ideally be maintained at a temperature conducive to metabolic activity. The shell of the body usually has a much lower mean temperature than the core, so that a thermal gradient exists from core to skin. The body's thermoregulatory system attempts to maintain this constant thermal gradient, despite the loss or absorption of heat at the surface (Fischer & Solomon, 1965, Wise, 1973).

The cutaneous responses to moderate localised temperature change, serve to assist body temperature regulation by vasoconstricting to cold stimuli, and vasodilating to warm ones. If the thermal stimulus is extreme and potentially damaging, the reaction serves to reduce the hazard, often at the expense of thermal economy (Hellon, 1963).

From studies completed on the circulatory responses to local heating, it appears that the increase in flow is confined entirely to the skin (Fox & Edholm, 1963, Hellon, 1963). Roddie (1983), postulates that the increase in forearm skin blood flow with local heating, occurs in two phases. The first phase is only a small percentage of the total circulatory increase, but it does involve a rapid change. He attributes this increase to the release of vasoconstrictor tone. The more prolonged second phase, coincides with the onset of sweating and bradykinin production, thought to be part of the sympathetic vasodilator mechanism.

There appears to be three main mechanisms for circulatory responses to heating. The first mechanism of decreased vasoconstrictor tone, relates to a reduced smooth muscle responsiveness, caused by the direct effects of heat on the smooth muscle cell. The second mechanism involves the cutaneous thermoreceptors. Cold receptors respond to warming by inhibiting their discharge, and the warm receptors by a proportionate increase in discharge rate (Downey, 1964). This altered neural output initiated by the thermoreceptors, will either result in a reduced adrenergic activity causing a further

release of vasoconstrictor tone, or an increase in cholinergic activity and the vasodilator mechanisms. Similiar changes are brought about by an increased spinal cord and/or hypothalamus temperature, the third controlling mechanisms.

The hypothalamus, is in fact the main thermoregulation center for responses to warm and cold stimuli. The preoptic and anterior portion of the hypothalamus are predominantly warmth sensitive, with the spinal cord and the peripheral skin receptors being the most sensitive to cooling. All of this information is thought to be integrated in the posterior hypothalamic region (Wise, 1973, Guyton 1976, Hellon, 1983).

The reaction of the skin blood vessels to cold is more complex, involving a number of factors which are often additive in their effects, and sometimes counteract each other (Folkow et al, 1963). The vessels of the skin respond to moderate local cooling with a rapid and persistent vasoconstriction. It appears that there are at least five mechanisms in operation to cause the vasoconstriction of the cutaneous vascular bed. The initial reaction is caused by the direct effect of cold on the smooth muscle cells, causing a contraction and constriction of the vessels (Folkow et al, 1963, Keatinge & Harman, 1980). Superimposed on this, are the reflex excitations of the vasoconstrictor fibers. The stimulation of cold receptors has a vasoconstrictor effect through various pathways. Cold and warm receptors are distributed throughout the skin, with the cold receptors being more numerous, (6-8 times more than warm receptors), and more superficial (0.1 mm compared with warm receptors at a depth of 0.3 - 0.6 mm) (Newburgh, 1968, Wise, 1973). This may account for the more pronounced cold response.

Afferent nerves from cold receptors, form axon reflex arcs which precipitate an immediate vasoconstriction of superficial blood vessels. The afferents pass on into the spinal cord, where some travel for one to two segments, and synapse with sympathetic nerves. Other afferents ascend all of the way to the thermoregulatory center via the posterior hypothalamus, to exert their influence (Downey, 1964, Wise, 1973). All three of these pathways originate with the cutaneous cold receptors. The final mechanism, involves the lowered temperature blood, reaching central thermoreceptors and evoking a

prolonged vasoconstriction. These mechanisms tend to operate if the cold stimulus is not prolonged (less than 10 min.), and does not cause the tissue temperature to fall below 12 °C.

Severe cooling, lowering the tissue temperature below 12 °C, usually elicits a reflex vasodilation which presumably prevents thermal damage (Keatinge & Harman 1980). Such a reflex dilation was first documented by Lewis (1930), and is termed a "Lewis reaction" or "hunting response". Lewis' explanation of the hunting response, was that if cooling was sufficient to damage the skin, it would provoke the release of "H substance". With time, the concentration of H substance rises to a level which is sufficient to produce a vasodilation, counteracting the vasoconstrictor effects of cold upon the vessels. As the blood flow to the area increases, the finger warms, the H substance is washed away, and the vasodilation weakens. Eventually vasoconstrictor tone predominates again, and the cycle repeats itself (Lewis, 1930).

Other mechanisms have been postulated which would account for the observed cold induced vasodilation. The hunting response appears to be mainly locally mediated (Duff et al, 1953, Sparks, 1978). Sparks (1978), proposes that the smooth muscle cell temperature falls so low, that the contractile mechanisms fail, causing a reduced constrictor effect. Added to this are, a cold induced insensitivity to adrenergic vasoconstrictor substances, vasodilatory axon reflexes from afferent pain fibers, and the accumulation of vasodilator metabolites (Folkow et al, 1963, Lee & Warren, 1978). These metabolites are presumably formed at the onset of thermal injury, in the cutaneous layers.

The phenomenom of cold induced vasodilation is confined to certain areas of the body. Fox and Wyatt (1962), using an "ice calorimeter", mapped the distribution of areas capable of producing a hunting reaction. These areas tended to be portions of the skin which were likely to be exposed to severe local cooling in cold climates. Such areas were also likely to contain numerous arterio-venous anastamoses, thought to be essential for a hunting reaction to occur, as pointed out by Kalenak et al (1975).

There is some question as to the existence of arterio-venous anastamoses in non-acral skin. Roddie (1983), states that there is no evidence of a hunting reaction occurring in forearm skin, as no arterio-venous anastamoses are present. However Fox and Wyatt (1962), noted a clear positive vasodilatory response on the forearm extensor surface of one of their subjects.

Barcroft and Edholm (1946) monitored deep muscle and subcutaneous temperatures of the forearm immersed in cold water (12 °C). The results revealed no oscillations in temperature over the 90 min. of measurement. Knight et al (1980), re-examined Lewis' theory by immersing fingers and ankles in cold water (1.4 °C). While the fingers behaved as Lewis described, they found no oscillations in ankle temperature with time.

It appears that the cold induced vasodilation response may be altered by prior exposures to cold. Nelms and Saper (1962), provided evidence of an enhanced cold induced vasodilation in the hands of fish filleters, in response to repeated cold exposures. The response became greater and occurred sooner in acclimatised body parts. Adams and Smith (1962), examined the cold induced vasodilation of cold conditioned fingers. They observed that the cold conditioned digits exhibited a shorter time between immersion and rewarming, a more rapid rewarming, and a higher final rewarmed temperature. The pain associated with cold immersions was diminished with conditioning and eventually disappeared completely. Adams and Smith, noticed that the unconditioned contralateral digits had a normal cyclic response to cold, indicating that the change in cold induced vasodilation occurred locally.

A reflex vasodilation after the removal of cold, has also been reported (Moore et al, 1966, Edwards, 1971). This after effect vasodilation has been observed as a temperature rise above the adjacent untreated body parts. The temperature reached a maximum after 15-20 min. (finger), and subsided three times as slowly (Lewis, 1930, Wise, 1973).

To suggest that cold vasodilation will occur in all body parts, would be an overextrapolation, as the reaction has only been observed in acral regions containing abundant arterio-venous anastamoses.

B. Skeletal Muscle

Circulation

Voluntary muscle has a rich blood supply derived from branches of neighbouring arteries. The number of supplying arteries is variable according to the muscle group, but does not appear to affect the volume of blood flow. The muscles of the forearm area are attributed with many arterial sources of blood supply (Hudlicka, 1973).

Walder (1968), claims that skeletal muscle has three circulatory pathways, each separately controlled. One pathway, accounting for a small percentage of the total muscle blood flow, nourishes the connective tissue of muscle, and the other two supply the metabolic needs of the muscle fibers. Hudlicka (1973), recognises a fast component of muscle circulation, nourishing the actual fibers, and a slow component serving the septa and tendons.

The arteries supplying a muscle, branch and anastamose freely with other branches, to form a primary arterial network. Small arteries are given off at regular intervals, running parallel to the muscle fibers, and anastamosing to form a secondary cubical network. Arterioles branch off regularly from these structures and run at right angles or obliquely to the muscle fibers. The terminal vascular bed is formed by these arterioles (with several layers of smooth muscle coat), branching into terminal arterioles (single layer of smooth muscle coat). These eventually split into capillaries, and at these branching points, smooth muscle cells are positioned in a circular fashion to form precapillary sphincters. Distal to the sphincters, a discontinuous helically arranged layer of smooth muscle tapers off to form the capillaries. These capillaries run longitudinally between fibers, and by frequent anastamoses, form a fine capillary network with oblong meshes. The capillaries eventually unite into venules, intercalcated between the arterioles. The venous system then reproduces and follows almost exactly, the arterial route (Walls, 1960, Barcroft, 1963, Hudlicka, 1973, Sparks, 1978).

Within muscle there are two main fiber types, the slow tonic fibers, which are red and have a high potential for aerobic metabolism, and the fast phasic fibers. These phasic fibers are generally white and have a higher anaerobic potential. Although there is not a strict

borderline between the two types of fiber within a muscle, phasic fibers constitute the bulk of the forearm muscle according to Folkow & Neil (1971).

A difference in the vasculature of the two fiber types has been noted, presumably due to differences in metabolic requirements. The capillaries to the phasic fibers form elongated loops which parallel the muscle fiber. Transverse branches arise from these, and encircle the individual fibers, forming a loose network around them. Tonic fibers are vascularised by capillaries forming shorter loops of equal length and breadth. This capillary network tends to be richer with a greater surface area, and closer contact with the individual fibers (Hudlicka, 1973). For both fiber types there is approximately one capillary to every fiber. This amounts to 3-4 capillaries around each fiber. As the phasic fibers are larger, they tend to have fewer capillaries in close proximity. Arterio-venous anastamoses which control large blood volumes in the skin, do not appear to be common in skeletal muscle. If they exist at all, it is probably in the septa or tendons (Hudlicka, 1973), or only on the surface of muscle (Walder, 1968, Sparks, 1978). Any control of blood in the capillary bed of muscle is probably through the functions of precapillary sphincters.

Control of Circulation

The muscle vascular bed consists of three distinct segments, which differ in their ability to regulate blood flow. The first of these segments contains the resistance vessels, which includes arterioles, metarterioles, terminal arterioles and precapillary sphincters. Resistance vessels are subject to considerable myogenic tone (Hudlicka, 1980), and this basal tone results from the vascular smooth muscle's response to passive stretch. The myogenic tone maintains a base level of vascular constriction in the vessels at rest (Little, 1981). These resistance vessels are also under vasomotor control, receiving sympathetic vasodilator and vasoconstrictor innervation (Guyton, 1976). The influence of these fibers is not substantial at rest, as a sympathetic block will only increase muscle blood flow by one fifth of it's maximum potential (Sparks, 1978). The vasoconstrictor fibers appear to influence most vessels in the vascular bed, but the vasodilatory fibers seem to act only on the precapillary segments,

most likely the arterioles (Bard, 1968, Hudlicka, 1980).

The smooth muscle of the arterioles and precapillary sphincters are sensitive to various metabolites, which generally cause a relaxation and increased blood flow. The transmural pressure of the resistance vessels and the tone of surrounding skeletal muscle may also affect resistance segment circulation (Hudlicka, 1980). Regulation of the resistance segment of circulation is primarily through a combination of myogenic and metabolic mechanisms, with metabolic influence being predominant when the ratio of metabolism to blood flow is high (Sparks, 1978). The second portion of the vascular bed involves the metabolic exchanges between the blood vessels and the extracellular spaces. Capillaries are the main vessels in this area, with muscle capillary beds differing from most other organ vascular beds, in that they have very few open capillaries at rest (Hudlicka, 1980). It is assumed that the precapillary sphincters are closed at rest, and an accumulation of metabolites would cause these to relax increasing blood flow to the area.

Capillaries and precapillary sphincters are thought not to have any direct vasomotor innervation. Metabolic requirements will normally determine blood flow to the area (Hudlicka, 1980).

Capacitance vessels constitute the final segment of the muscle vascular bed. These vessels function to enable shifts and storage of blood volume without altering the blood pressure. Capacitance vessels are in fact the venules and veins, possessing smooth muscle coats of the multiunit type. This smooth muscle has no tonic pacemaker activity, and the vessels consequently have no significant basal myogenic tone (Little, 1981). These vessels therefore require extrinsic factors to provide vasomotor activity. Skeletal muscle veins are well supplied with alpha receptors, but have very few beta receptors (Allwood et al, 1963, Sparks, 1978). Veins are therefore well supplied with vasoconstrictor influences, but have no real vasodilator innervation.

To summarise, at rest the skeletal muscle vascular bed is controlled by a significant basal myogenic tone, vasoconstrictor tone, and smooth muscle responses to various stimuli.

Factors Influencing Blood Flow

At rest the skeletal muscle blood flow ranges from 4 - 7 ml./100 ml. of tissue/min., and remains relatively stable, except in response to exercise. Other events and stimuli do not appear to have as dramatic, or as prolonged effects on muscle blood flow. These include:

1. Exercise

There is an increase in muscle blood flow accompanying muscular contraction. The change is thought to be mediated by metabolic substances formed by the active muscles, acting locally on the resistance vessels (Shepherd, 1983). Roddie et al (1963), suggest that the non exercising muscles receive increased vasoconstrictor tone, to aid in redistributing blood to the exercising muscles.

2. Emotional Stress

Blood is redistributed throughout the body in response to emotional stress. As described above, certain skin vessels may vasoconstrict in reaction to stress, but skeletal muscle vessels will increase their blood flow. This increase is believed to be mediated by an increase in vasodilator activity (Roddie, 1963, Downey, 1964, Vander et al, 1980). Acetylcholine, histamine and another unidentified vasodilator mediate the vasodilation (Sparks, 1978). According to Guyton (1976), the vasodilator fibers are activated by a special nervous pathway beginning in the cerebral cortex. Noxious stimuli may also increase muscle blood flow, but in this case, the nociceptors mediate an adrenaline response, stimulating the beta receptors on the arterioles (Hudlicka, 1980). Bard (1968), suggests that the anticipation of exercise may elicit an early increase in blood flow, although this theory has not been substantiated elsewhere.

3. Changes in Pressure

In contrast to the cutaneous circulation, the skeletal muscle blood flow is sensitive to sudden changes in posture and perfusion pressure. In response to a head downward position, or elevation of the legs, the forearm blood flow increases as a result of reduced vasoconstrictor tone. The forearm blood flow decreases with a shift from a horizontal to

vertical position, and this is a result of increased vasoconstrictor tone. The baroreceptors appear to alter blood flow by altering the vasoconstrictor tone only, to maintain suitable perfusion pressure (Roddie et al, 1963, Hudlicka, 1980).

4. Temperature

It is suggested by Shepherd (1963), that the thermoregulatory center utilises only the vasomotor fibers to the skin, and muscle blood flow control plays only a minor part. It therefore appears that sympathetic fibers to muscles are not influenced by changes in body temperature (Clement, 1979). This has been confirmed by experiments measuring the oxygen saturation of blood from superficial and deep veins, which have established that the circulatory increase in response to body heating is entirely confined to the skin (Roddie et al, 1963, Detry et al, 1972). With respect to cold stimuli, there is no good evidence for a reflex constriction in the vessels of the forearm muscles, in response to local or direct cooling (Fox, 1961, Downey, 1964). Direct cooling of the muscle blood vessels will cause the smooth muscle to constrict and reduce blood flow (Keatinge & Harman, 1980), but the thermal gradient between the muscle and the skin surface reduces the possibility of any direct cooling effects

Keatinge (1961), observed a vasodilation in the deep vessels when the finger was cooled. He suggested that this could be the result of an impaired response to constrictor hormones, a reflex related to the vasoconstriction of skin vessels, or an effect initiated by a central controlling mechanism (Haines, 1967, Keatinge & Harman, 1980). These theories may be tenable, considering Bard's (1968) finding, that when the skeletal muscle vasodilator fibers are stimulated electrically in the hypothalamus, an accompanying vasoconstrictor discharge to the skin helps to redistribute cardiac output.

Muscle blood flow in the resting state, is far more stable than the cutaneous circulation. If temperature has any reflex effect on muscle circulation, it is possibly to countereffect the changes in the cutaneous circulation.

C. Measurement of Blood Flow

Introduction

Blood flow measurement is possible by a variety of methods. Thermal flowmetry, electromagnetic flowmeters, radioactive tracer clearance, ultrasonic flowmeters, and plethysmography are examples of the available techniques, which often differ in validity and application.

Venous occlusion plethysmography, which involves the measurement of volume changes, was first adapted for limb blood flow measurement by Hewlett and van Zwaluwenberg (1909). Since then it has been used extensively for non-invasive measurement of peripheral blood flow. Rushmer, Baker, and Stegall (1966) state, that if blood flow is defined as the volume change per unit time, then the most direct measurement approach should involve techniques sensitive to volume change. They consequently rate plethysmography highly for it's ability to; measure the target variable (blood flow), it's freedom from unrelated extraneous variables, it's freedom from measurement induced influences, it's stability, sensitivity, and range of measurement.

Plethysmography employs an occluding cuff, applied to a limb and inflatable to a sub-diastolic pressure. If the pressure used is sufficient, it causes venous closure and occludes outflow from the limb. The subsequent increase in the volume of the limb distal to the occluding cuff, is equal to the arterial inflow during the recording period.

Whitney (1953), adapted the mercury in rubber strain gauge for the plethysmographic estimation of limb blood flow. The elasticity and compliance of the mercury in rubber strain gauge, means that it is particularly suitable for the measurement of girth changes, of asymmetrically shaped limbs.

Mercury Strain Gauge Plethysmography

Theoretical Basis

The mercury strain gauge has high sensitivity, and for small changes in gauge extension, the change in resistance is essentially linearly related to the change in length, however it's low resistance, makes it technically difficult to use.

Unlike volume plethysmography, strain gauge plethysmography does not record the direct volume changes, but measures changes in the circumference of the limb segment which it encircles (Englund et al, 1972). This method assumes that human limbs and digits, approximate a circular cross section (Whitney, 1953). In fact limbs tend to be elliptical in cross section. with plethysmography tending to overestimate blood flow (Whitney, 1953, Englund et al, 1972). Venous occlusion plethysmography is based on the observation, that for small changes in the volume of a cylinder of constant length, the volume change is approximately twice the change of it's circumference (Levy et al, 1979).

Two main assumptions are made for strain gauge plethysmography. The first is that changes in limb volume are only due to radial expansion, and there is no change in the dimension measured along the long axis of a limb. Radial isotropy is the second assumption. This considers that circumference changes must be due to changes in all transverse directions (Whitney, 1953, Levy et al, 1979).

Although the theoretical basis for strain gauge plethysmography has been challenged (Ardill et al, 1968), it is generally considered to be a valid method of blood flow measurement for work involving repeated comparisons (Knight & Londeree, 1980). Sparks (1978), claims that venous occlusion plethysmography is widely accepted as the standard method of skin blood flow measurement in man. The Whitney strain gauge is probably the most practical form of plethysmography, and is used as the standard for blood flow measurement at the NASA physiological performance laboratory (Levitan et al, 1983).

For venous occlusion plethysmography, mercury strain gauges are employed as length transducers, registering the changes in circumference. The gauge consists of a fine

bore of rubber (or silastic) tubing filled with a column of mercury and plugged with conducting endpieces, which are soldered to connecting wires. The mercury in rubber strain gauge forms one arm of a Wheatstone bridge circuit, with the adjacent arm in series, being a potentiometer, and the other resistances in parallel occupied by high stability/low value resistances.

If the gauge is stretched, the diameter of the mercury thread is reduced and the gauge resistance increases. Deflections of the recording stylus, therefore provide a measurement of changes in the strain gauge resistance. For small changes in length (circumference), the resistance change is linearly related to the change in the gauge length (Whitney, 1953, Needham, 1972).

If a cylinder expands only slightly in a radial direction, the radius changes from r to $r + z$.

Therefore the change in cross sectional area of the cylinder

$$= \pi(r+z)^2 - \pi r^2$$

$$= 2\pi rz, \text{ if } z^2 \text{ is small.}$$

The percentage change in area is given as;

$$= \frac{2\pi rz}{\pi r^2} \times 100$$

$$= \frac{2z}{r} \times 100$$

The change in the circumference of the cylinder is;

$$= 2\pi(r+z) - 2\pi r$$

$$= 2\pi z$$

And the percentage change in circumference is provided by;

$$= \frac{2\pi z}{2\pi r} \times 100$$

$$= \frac{z}{r}$$

Thus the percentage change of circumference equals one half of the percentage change of area (volume) (Clarke & Hellon, 1957)

Validity of Technique

Several studies have examined the validity of mercury in rubber strain gauge plethysmography . Clarke and Hellon (1957), compared mercury strain gauge plethysmography on one hand, with water plethysmography on the contralateral limb. They noted good agreement between the two methods, but the water plethysmography consistently gave an average of 9.4% higher blood flow readings than the strain gauge. Burger, Horeman and Brakkee (1959), using water filled plethysmography, found that this method gave an average of 17% higher blood flow than that indicated using the mercury strain gauge technique. Dahn and Hallböök (1970), made simultaneous blood flow measurements on the lower leg, using water plethysmography and four mercury in rubber strain gauges at equal intervals along the limb. Recordings of flow were made during reactive hyperemia, with the two middle strain gauges and the water plethysmograph yielding practically identical blood flow recordings. The most proximal of the strain gauges, gave values that were always higher than the water plethysmograph, and the distal recordings were consistently lower. Longhurst and co-workers (1974), simultaneously measured forearm blood flow with a single strand mercury strain gauge, and brachial artery flow with an electromagnetic flowmeter. The two measurements had a correlation coefficient of 0.823, with the strain gauge plethysmography tending to give higher blood flow values.

Englund, Hallböök and Ling (1972), using extracorporeal perfusion, found that there was a linear relationship between actual flow (supplied by a heart-lung machine), and mercury strain gauge recordings. Levy (1979), compared mercury strain gauge plethysmography, water plethysmography, and pulsed doppler flow measurements. He

concluded that while strain gauge plethysmography was not quite as sensitive as the water method, it was more practical in it's applications. Mercury strain gauge plethysmography was compared with ultrasound and impedance plethysmography, by Levitan et al (1983). Although differences were observed in the magnitude of blood flow estimation, the magnitude of the changes in flow were tracked identically by all three methods. Mercury strain gauge plethysmography may lack some validity, in that it tends to underestimate actual blood flow, but it appears to be extremely reliable. It is particularly suitable for repeated measurements of blood flow, and will accurately reflect the changes in flow that occur.

Temperature Effects on Strain Gauge

The thermal coeffficient of resistance is relatively high for mercury, so that a 1 °C change in temperature, is approximately equal to the resistance change produced by a 0.05% change in the gauge length (Whitney, 1953). Because of thermal instability, Whitney (1953) suggests winding copper wire around the strain gauge, and linking it to an adjacent arm of the Wheatstone bridge, to compensate for temperature effects on the resistance. Honda (1962), submits that resistance changes with temperature are of no consequence when pulse volume or blood flow are being monitored. They can be a problem, he states, when temperature induced changes in volume are being investigated. Brengelmann, Wyss and Rowell (1973), found temperature compensation to be unnecessary. They conducted a study using a hollow copper "arm", through which different temperatures of water could be circulated. For a 10 °C temperature change, a resistance increase equivalent to a 1 mm. increase in length was observed. They concluded that the accuracy of monitoring changes in gauge length, was insignificantly affected by a 10 °C temperature change.

Hydrostatic Effects

The effect of hydrostatic pressure on venous occlusion plethysmography has been considered by researchers. Wallace (1958), measured forearm blood flow by water plethysmography. Normally the height of the water above the forearm was between 9 and 16 cm. For twelve subjects this water level was increased to 16 to 26 cm. above the forearm. Some evidence of reduced blood flow was noticed in this range, but it was deemed negligible by the researcher.

Dahn and Hallböök (1970), with their comparison of blood flow measurements by air, water, and mercury strain gauge plethysmography, noted that the hydrostatic pressure tended to reduce limb blood flow, and accelerate venous emptying after occlusion. The differences were again small and insignificant. A comparative study of water and mercury strain gauge plethysmography, was carried out by Francis and McCaig (1985), to examine the hydrostatic effect on foot blood flow. They found that foot blood flow was inversely proportional to the depth of water in the plethysmograph. These writers proposed three possible explanations for such changes. Firstly, they suggested that a small leak with venous occlusion, occurred via unoccluded interosseous veins, which while insignificant when the plethysmograph was empty, became more obvious with the hydrostatic pressure effect. They also submit, that hydrostatic pressure reduces the pressure differential between the aorta and the foot, resulting in preferential blood supply to other areas. The third and most plausible explanation advanced by Francis and McCaig, is that the hyrostatic pressure on the ankle (supine lying with the foot perpindicular at 90 °), would partially occlude venous return at the ankle. This would cause the distal capillary beds to partially fill, resulting in a reduced capacity to accept blood during venous occlusion. Because the forearm is horizontal during plethysmography this partial venous occlusion does not occur to the same degree, and the hydrostatic effect is less significant (Francis and McCaig, 1985).

Calibrations

The first method of strain gauge calibration suggested by Whitney (1953), has subsequently been criticised. He attached the strain gauge to a perspex mount, and suspended a 25 g weight from the doubled strand. For this degree of gauge tension, a pen deflection was obtained. He then mounted the gauge on a cylindrical form, and using the tension adjusting nut, reproduced a tension of 25 g. A measurement of the gauge length was made in this position, and calibration was carried out by varying the gauge length by known amounts. Hallböök, Mansson and Nilsen (1970), claimed that the gauge mounting device causes discrepancies, as it does not allow the gauge to completely encircle the limb, and tends to hold the ends of the gauge up off the limb. They propose a constant current calibration device, which permits in vivo calibration. The device induces a voltage difference across the output, which produces a stylus deflection equivalent to a one per mille volume change. Brengelmann, Wyss and Rowell (1973), modified Whitney's calibration method, by using a 25 g tension and measuring deviations from the baseline by stretching the gauge by known amounts. However instead of using the gauge to monitor the circumference of the limb continuously, they simply measured the limb circumference before and after the gauge application. The average of these two girths was used as the limb circumference throughout recording.

Occlusion

With venous-occlusion plethysmography, the method of occlusion is critical. Although the occlusion pressures used are often given in reports, details on cuff size are often not included. Investigations into the optimal cuff size for blood pressure measurement (Geddes, 1970), have indicated that the accuracy of blood pressure readings varies with different cuff widths. Presumably the occlusion pressure differs with the length of the vascular segment occluded. The wider the occluding cuff used, the less the occluding pressure required to collapse the underlying venous segments. Most studies appear to have used standard blood pressure occlusion cuffs (approximately 12 cm in width), although

the occlusion pressure tends to vary. Cooper, Edholm and Mottram (1955), inflated their forearm occlusion cuff to 70 mm Hg. Johnson, Rowell and Brengelmann (1974), used an occlusion pressure of 40 mm Hg for forearm blood flow measurements. Dahn and Hallböök (1970), investigating calf blood flow, used a venous occlusion pressure of 50 mm Hg. The occlusion pressures used, will also tend to cause the capillaries, arterio-venous anastamoses, and arterioles, originating from the arterial branches proximal to the cuff, to collapse (Burch, 1954). Burch (1954), claims that while the quantity of blood obstructed from flowing through these small branches is not known, it probably represents a small percentage of the total flow.

Ideally a systematic investigation of each subject, to establish the best cuff pressure, may be necessary to give a constant blood flow measurement, as suggested by Christensen and Nielsen (1942). Ideally the occluding pressure will be sub-diastolic, to ensure that arterial inflow continues unimpeded, but will be sufficient to occlude venous return.

The occlusion period is also an important factor in plethysmography. Sparks (1978), states that the occlusion periods must be brief to avoid raising the venous pressure to a point where it will retard arterial inflow, or cause leakage of blood past the occluding cuff (Kitchin, 1963). Ideally the occlusion time should be sufficient to obtain a suitable plethysmographic trace, from which a tangent can be drawn. A cuff inflation artifact usually occurs at the beginning of each trace. This artifact corresponds with the deformation of the limb tissues, towards the strain gauge, by the occluding cuff (Christensen & Nielsen, 1942). Sparks (1978), suggests a brief occlusion period of approximately 5 sec., which appears to be standard (Cooper et al, 1955, Dahn & Hallböök, 1970).

It is claimed that if the limb is not above heart level during recording, the veins will not be able to accomodate the inflowing blood (Greenfield et al, 1963, Rushmer et al, 1966). Christensen and Nielsen (1942), recommend that the limb should be 20 to 30

cm. above heart level, or no filling of the veins will occur, and accurate blood flow measurement will not be possible. It is generally recommended that the limb should be at least level with the heart, for the most accurate blood flow determinations (Cooper et al, 1955, Eagan, 1961).

It would appear that some form of hand blood flow occlusion should be made in order to obtain accurate flow measurements on the forearm. Acral body parts demonstrate fluctuations of blood flow, in response to a multitude of environmental stimuli. because of the lability of the hand circulation, several investigators (Kerslake, 1949, Cooper et al, 1955, Johnson et al, 1974), have occluded hand blood flow while measuring forearm circulation. Quinn (1965), measured forearm blood flow with water plethysmography. He noted that a wrist cuff inflated to 200 mm Hg, gave average apparent flows 19% lower than when no wrist cuffs were used. Kerslake (1949), occluded hand blood flow with a wrist cuff inflated to between 230 and 240 mm Hg. Forearm blood flow was found to initially increase after occlusion, followed by a decrease, with flow eventually stabilising at an intermediate level after approximately 1 min. Cooper, Edholm and Mottram (1955), used a wrist cuff inflated to 200 mm Hg, while monitoring forearm blood flow. Johnson, Wyss and Rowell (1974), also utilised a wrist occlusion cuff, which they inflated to 280 mm Hg for 4 out of every 5 minutes.

Forearm Blood Flow Measurement

Blood flow measurements are frequently made on the human forearm. It lends itself to such investigations, because of it's ability to be raised above heart level, and positioned in water baths and plethysmographs. The proximal portion of the forearm is almost circular in cross section, particularly if the forearm is pronated. The distal segment is more elliptical in cross section. Proximally the radius and ulna are positioned relatively deep in the forearm, ensuring a more or less radial expansion of the soft tissues in all directions. As the radius and ulna are more superficial collaterally in the distal portion, any expansion will presumably be

in the volar and dorsal directions.

Cooper, Edholm and Mottram (1955), dissected five cadaver forearm segments, corresponding with the section normally enclosed in a plethysmograph. The resulting average volume compositions were; skin 8.6%, bone 13.7%, tendon 6.1%, fat, fascia, nerves and blood vessels 8%, and muscle 63.6%.

Forearm blood flows measured, cover a wide range of values. Barcroft and Edholm (1946), using an air plethysmograph, estimated forearm blood flow to be 3.1 ± 0.25 ml/100 ml of tissue/min. Cooper, Edholm and Mottram (1955), with water plethysmography, provided values ranging from 1.45 to 10.5 ml/100 ml of tissue/min. Zelis, Mason and Braunwald (1969), calculated average forearm flow, by mercury strain gauge plethysmography, at 6.3 ± 0.58 ml/100 ml of forearm/min., and Mottram (1983), also using strain gauges, found a flow between 3.2 and 3.7 ml/100 ml of muscle/min. Some of these differences may be explained by Dahn and Hallböök's (1970), successful demonstration that the level of strain gauge application on the limb, often determines the blood flow recorded. They reasoned that the skin/muscle/bone composition varies along the length of the limb and will likely be reflected in the blood flows recorded at each site.

Clarke, Hellon and Lind (1958), attempted to partition skin and muscle blood flow measurements by monitoring flow with proximal and distal strain gauges. They suggested that the distal segment of the forearm is composed mainly of skin, bone and tendons, whereas the proximal section is predominantly muscle. In their study, the midpoint of the forearm was measured as halfway between the olecranon process and the ulnar styloid process. A proximal gauge encircled the forearm 6 cm above this point, and the other gauge 6 cm distal from the midpoint. After 40 min. of immersion in 1°C water, an increased blood flow, three to four times above resting, was noted. The distal gauge flow did not reach as high a level as the upper forearm. Adrenalin iontophoresis was used to stop forearm skin blood flow, and did not alter the blood flow pattern, confirming that the increased blood flow with cooling was confined to the muscle. From these experiments it appears that proximal and distal strain

gauges applied to the forearm, will reflect muscle and skin blood flow respectively. Proximal measures would include flows to muscle, tendon bone, fat and connective tissue. (Cooper et al, 1955). As tendon, connective tissue and fat are relatively avascular, and the bone blood flow is estimated as only 0.5 to 1.0 ml/100 ml of bone/min., most of the measured flow is thought to be to the actual muscle tissue (Cooper et al, 1955). Zelis, Mason and Braunwald (1969), have calculated that approximately 52 % of the total forearm blood flow is to the muscle tissue.

D. Cold Therapy

Sensations

The sensation of cold is felt when the temperature of a substance is lower than that of the body area to which it is applied. Cold application will evoke different sensations, depending on which part of the body it is applied to, and the form of cold which is utilised. Usually the first sensation will be one of cold, which progressively becomes more uncomfortable over a period of approximately 3 minutes. This is replaced by a burning sensation, lasting up to 5 minutes, and then an aching of approximately 1 minute. The fourth and final sensation is that of analgesia, in which there is no real sensibility to pain (Grant, 1964, Behnke, 1973, Kramer & Mendryk, 1979).

Hocutt (1981), has listed the cold sensations a little differently. Stage 1 consisted of a cold feeling from 1 to 3 minutes, stage 2 at 2 to 7 minutes, of burning and/or aching, and stage 3 at 5 to 12 minutes, of local numbness and anesthesia. The final stage, he states, occurs at 12 to 15 minutes, and consists of a reflex deep tissue vasodilation and warming. Bugaj (1975), observed that analgesia was achieved at an average of 1 minute and 45 seconds after ice massage was initiated, and disappeared approximately 3 minutes after the treatment was discontinued. He also noted that analgesia occurred only after the skin temperature was reduced to 13.6 °C.

Raptou (1968), claimed that ice massage produced slightly different sensation timing. For 1.5 to 2 minutes, a cycle of intense cold, burning and intense discomfort ensued. This was followed by anesthesia, analgesia and relaxation. This may have been due to the stimulation of mechanoreceptors with the massage technique, or it could be that the areas appropriate to ice massage (i.e. larger flatter body parts), react differently than the body parts which are normally immersed (i.e. hands, fingers and feet).

Penetrative Effect of Cold

The extent of the therapeutic effects of cold, is largely dependent on the temperature of the substance applied, the duration of application, and the region of the body to which it is applied.

The region of the body to which cold is applied determines how far the superficially applied cold penetrates, and how much the resting thermal gradient is disturbed. The thickness of the skin and the subcutaneous tissues, have been shown to affect the penetration of cold. Johnson et al (1979), reported a significant relationship between subcutaneous body fat and the reduction of intramuscular temperatures. Because of it's low thermal conductivity, subcutaneous fat forms a barrier to heat exchange (Lowdon & Moore, 1975). Because of this effect, short applications of cold do not tend to penetrate, and lower muscle temperature significantly (Lehmann et al, 1974). The penetrative effect of cold also depends on the water content of the tissues, and the rate of blood flow through the cooling area (Stangel, 1975).

Attempts to evaluate the penetrative effects of cold, have produced equivocal results. The temperature of the skin falls rapidly upon cold application. Decreases of 12 to 16 °C within the first two minutes, have been noted (Lee & Warren, 1978). By comparison, the deep muscle temperatures fall only gradually, and then by only small amounts (Waylonis, 1967, Olson & Stravino, 1972, Lee & Warren, 1978). The amount of cold penetration into the deep muscular tissues, appears to be questionable. De Lateur et al (1970), observed no temperature changes below 3.0 cm with 10 minutes of surface cooling at 7 °C. Bierman

(1939), using a thermocouple deep within the gastrocnemius muscle, cooled by ice bags on either side, found only a 3.5 °C drop in temperature after 2 hours and 10 minutes. Lowdon and Moore (1975), however, observed a large temperature drop (17.9 °C), 2 cm into the biceps brachii muscle, developed after only 5 minutes of ice massage.

Most of the evidence points to rather poor penetration of cold into the deep tissues, and certainly not to the extent which is observed in the skin. Because of the low thermal conductivity of subcutaneous fat, any deep cooling that does take place, tends to be maintained for extended periods of time (Laing et al, 1973, Lehmann et al 1974). Although the blood flow in muscle is reduced by the direct effects of cold on the vessels (Abdel-Sayed et al, 1970, Keatinge & Harman, 1980), it appears unlikely that cold will penetrate sufficiently to mediate any direct changes to the muscle circulation.

Duration of Cold Application

Many workers have apparently made recommendations based on Lewis' (1930) findings. He noticed that immersion of the index finger in a 5.4 °C water bath, caused an initial vasoconstriction lasting between 9 and 16 minutes, followed by a vasodilation lasting 4 to 5 minutes. This cycling of local blood flow was termed by Lewis, the "hunting response".

With acute injuries, cold application is timed to supposedly avoid or minimise the vasodilation that occurs with the hunting response. Wise (1973), suggests applications of 10 minutes interspersed with 10 minutes of no treatment, Hocutt (1981) prescribes 5 to 12 minutes of cold therapy, and Kramer and Mendryk (1979), suggest that cold should not be used for more than 20 to 30 minutes at any one time. Moore et al (1966),advise that in order to limit vasodilation of the deeper vessels, cryotherapy should be limited to 20 minutes maximum at any one time. These authors admit that the time frames are arbitrarily selected, as cycling varies with body areas.

Once injuries are beyond the inflammatory stages (48 hrs plus), cold application is prolonged in an attempt to evoke a reflex vasodilation, with the cold being removed before the

second vasoconstriction occurs (Chu & Lutt, 1969). Presumably the time required for these effects is slightly longer than for the application of cold to acute injuries, described above. Edwards (1971), suggests that between 30 and 60 minutes of cold therapy is sufficient to achieve the desired effects. In any case, the removal of the cold stimulus should provoke a reflex after effect vasodilation according to Moore et al (1966).

Physiological Effects of Cold

The beneficial and therapeutic physiological effects of cold are commonly recognised to be a reduced inflammmatory response, diminished pain from the injury, a decreased muscular spasm, and a reduction of hemmorrhage and blood flow at the injury site.

Inflammatory Response

Behnke (1974), has classified athletic soft tissue injuries into four distinct stages; the trauma itself, hematoma formation, the subsequent hematoma reabsorption, and healing by scar tissue. Two of the major limiting factors in athletic injury rehabilitation, are the extent of hematoma formation and the expediency of that hematoma reabsorption.

With sport's injuries, damage and rupture of vascular structures is likely, allowing direct hemmorrhaging into the interstitial spaces. In addition to this, local inflammatory responses occur, which tend to exaggerate the edema and eventual hematoma formation.

When a vessel is damaged, the arterioles, capillaries and venules in the proximity, undergo a short lived vasoconstriction, lasting from 10 seconds to 2 or 3 minutes (Lee & Warren, 1978). This transitory slowing of the circulation allows leucocytes to move out of the capillaries by diapedesis. These cells usually engulf and phagocytise irritants, but because few irritants are present in athletic injuries, they are often superfluous, and merely add to the cellular debris (Wise, 1973, Knight, 1976).

The injured tissues release various substances, such as serotonin, bradykinin and histamine. These compounds are often referred to as "H substances" by certain authors (Lewis, 1930, Wise, 1973), said to be due to their histamine-like action and their uncertain identification (Wise, 1973, Lee & Warren, 1978). These substances generally cause a vasodilation in the precapillary beds, and a vasoconstriction in the postcapillary segments (Lee & Warren, 1978), with a resulting net increase of blood flow into the traumatised area, and a decreased flow out of this area. The vasodilation may increase blood flow to the area by as much as tenfold, and is accompanied by a change in the vascular permeability (Lee & Warren, 1978).

The increased capillary blood flow means that the capillary hydrostatic pressure is increased, precipitating a movement of fluid out of the capillaries into the interstitium. An increased permeability of the small blood vessels in the injured area also occurs, probably as a result of the endothelial cells pulling apart. This permits plasma proteins and colloids to move out into the interstitium, causing an elevated colloid osmotic pressure, which tends to draw further fluids out of the capillaries (Knight, 1976, Lee & Warren, 1978).

Because of the reduced blood flow through the traumatised area, many cells suffer secondary hypoxic damage, causing a breakdown of the cell and release of further substances capable of increasing vessel permeability. This adds to the hydration of the intercellular spaces and edema formation (Knight, 1976).

The overall inflammatory response is considered by Wise (1973), to be an overreaction, relative to the damage caused by an athletic injury. The secondary inflammatory reactions result in additional edema, which combined with the direct hemmorrhaging, forms an excessively large hematoma. This extravascular fluid tends to be thicker than most serous fluids, causing the lymphatic system to become clogged. Hematoma reabsorption is restricted by this stagnation of fluid, and the stage is set for adhesion formation (Kramer, 1977, Mason, 1978, Knight, 1982).

As described above, the recovery time from an athletic injury is often determined by the size of the hematoma formation, and the speed with which the body is able to mobilise the injury debris (Barnes, 1979). Any retardation of the initial inflammatory reaction will limit the hematoma size and ensure an earlier return to activity. Cold applications are widely prescribed for reducing the inflammatory response of athletic injuries, and appear to have several effects on the reactions taking place.

Janssen and Waaler (1967), demonstrated that cold temperatures reduced the degree of inflammation, the capillary permeability, and the general cellular response to irritative trauma. Farry et al (1980), working with traumatised pig ligaments, found that the inflammatory reaction, including biopsied evidence of edema, was reduced in the cold treated ligaments.

An obvious effect of cooling is a reduction of the metabolic requirements of the injured area and surrounding tissues (Olson & Stravino, 1972). This cooled environment, reduces the oxygen needs of the tissues, causing fewer metabolic by-products to be formed (Kramer & Mendryk, 1979), and limiting the secondary hypoxic damage.

Cold also limits edema formation by reducing the hydration of the intercellular spaces. Olson and Stravino (1972), claim that cold initially provokes a direct and persistent constriction of the superficial blood vessels, a reflex vasoconstriction via the central nervous system, and a delayed vasoconstriction from hypothalamic activation. From the previous sections on muscular and cutaneous circulations, it must be assumed that these reactions are confined to the cutaneous circulation.

Cold appears to limit edema formation by a variety of mechanisms. The viscosity of blood increases with cooling, and along with the vasoconstriction, acts in slowing blood flow through the area. These two factors combined with the reduced capillary surface area, caused by the smooth muscle constriction, work to reduce capillary permeability and capillary hydrostatic pressure. This reduced hydrostatic pressure apparently favours the reabsorption of fluid from the interstitium, and restrains filtration out of the capillaries

(Kitchin, 1963, Stangel, 1975, Knight, 1976, Lee & Warren, 1978).

The mechanism of vasoconstriction, and a reduction of the local cellular metabolism, figure prominantly in limiting edema, and the formation of hematoma

Pain

Although pain does not interfere directly with the soft tissue healing processes, it is probably the most aggravating complication.

Cold has been demonstrated as an effective pain reliever. Benson and Copp (1974), produced evidence that 20 minutes of ice application was significantly more effective than 20 minutes of short wave diathermy, in elevating the pain threshold to pressure.

Several theories have been advanced on the mechanism of the pain relief which cold mediates. Chu and Lutt (1969), propose that cold numbs pain by causing the sensory nerve endings to conduct fewer impulses to the central nervous system. Laing et al (1973), suggest that it is the local tissue response to injury which causes the most discomfort. Cooling depresses these responses, which has a more significant effect on pain relief, than the reduced nerve conduction. The most popular explanation appears to be that of "counter irritation". It is claimed that cold stimulates the cutaneous receptors, causing a bombardment of the central pain receptors to such a degree, that afferent pain impulses are obliterated (Olson & Stravino, 1972, Wise, 1973, Stangel, 1975, Kramer, 1977).

Muscle Spasm

The application of cold has been found to be a very useful method of reducing the muscular spasm which is often associated with a soft tissue injury. Muscular spasm tends to cause pain through a "spasm-ischemia-pain-spasm" cycle, and also restricts early mobilisation of injured parts. Further secondary damage occurs as a consequence of muscular spasm. The vasoactive substances released by damaged vessels, combined with

the actual force of the injury, may cause irritation of nerve endings, resulting in pain. The pain often mediates muscular spasm, which also decreases blood flow through the area and forms additional vasoactive metabolites (Hocutt, 1981).

Boes (1962), recorded the electromyographic (EMG) activity of the patellar tendon reflex response in hemiplegics and quadriplegics. The magnitude of this response diminished by approximately 34% during cold pack application. Licht (1965), states that muscle spindle activity begins to become depressed at relatively high muscle temperatures (32 °C), so that a great deal of cooling is not required to reduce spasm. This reduction in muscle spindle activity with cooling, is one step towards reducing spasm, and allows movement of the area without eliciting the stretch reflex (Raptou, 1968).

Cooling also diminishes the rate of nervous impulse transmission. Nerve conduction begins to fail at 27 °C and is blocked at approximately 10 °C (Raptou, 1968). This along with the counter irritative effect of cold, breaks the pain reflex arcs, preventing efferent motor impulses to spastic muscles (Olson & Stravino, 1972). The final mechanism of spasm reduction, cited by Stangel (1975), is that cold alters the viscosity of muscle, reducing it's contractability by some degree. It is probably a combination of these mechanisms in conjunction with the pain reduction, which helps to break the muscle spasm cycle, and permit a more comfortable and speedier recovery.

Circulation

In interpreting their observations, therapists have extrapolated from Lewis' findings on the circulation to the finger-tip, confusing the physiological effects of cold on the circulation (Knight, 1976). There are several points about Lewis' (1930) study, which should be considered when extrapolating from his results.

Firstly, the measurements were conducted on a finger tip, a highly vascular area with numerous arterio-venous anastamoses, and not at all a typical body part (Grant & Bland, 1931). The findings of Fox and Wyatt (1962), have demonstrated that the responses of different cutaneous body sites, vary widely in the magnitude and type of

response to cold.

A second point, is that the finger has a high bone and skin composition, with very little underlying muscle. Blood flow in the finger tip is largely confined to the skin, so that extrapolations from these vascular changes observed, to the muscle circulation, may be erroneous.

The fact that Lewis measured finger temperature, and not finger blood flow, is a third consideration. According to Greenfield (1963), the temperature of the skin depends on so many other factors, that it is a very imperfect index of skin circulation. Skin temperature depends on; the temperature of arterial blood, the rate of blood flow through distal and subjacent parts, the nearby muscular activity, the rate of sweat evaporation, and the temperature, humidity, pressure, and motion of the air (Greenfield, 1963).

It is common for cryotherapy exponents to extrapolate from the vascular changes noted in the fingers by Lewis (1930), to circulatory changes in other vascular beds. These misinterpretations are often not apparent, until actual blood flow measurements are made.

Folkow et al (1963), enclosed one of a subject's hands in a water plethysmograph, with the water temperature at 0 °C. The contralateral hand was placed in a similiar plethysmograph with the water at 35 °C. Blood flow in the experimental hand decreased from 15 ml/100 ml of tissue/min. to 1-2 ml/100 ml of tissue/min. Control hand blood flow was also reduced, but less markedly. This vasoconstriction disappeared within 3 to 4 minutes, whereas the experimental effect remained intense for between 6 and 15 minutes. The researchers postulated that a reflex neurogenic component reduced the blood flow in both hands, but the experimental hand, had a local cold induced vaso-constriction superimposed to produce the larger flow reduction.

Knight and Londeree (1980), measured the blood flow to the ankle, using strain gauge plethysmography. They compared 25 minutes of local heating (67 °C towels), and 25 minutes of local cooling (-7 °C cold packs), with controls. Blood flow in the cold

treated ankle was significantly reduced, and remained at this level for the 20 minutes of post-treatment monitoring.

Clarke, Hellon and Lind (1958), utilised strain gauge plethysmography to measure forearm blood flow under different conditions. These experimenters observed little change in forearm blood flow in 10 °C water baths, a slight increase at 6 °C, and a pronounced increase at 1 °C. This increase was sometimes up to 3 or 4 times the resting blood flow. The use of upper and lower strain gauges, and adrenaline iontophoresis, confirmed that the observed flow increase, was mainly confined to the muscle circulation. Clarke and his co-workers, suggested that this forearm vasodilation was caused either by the direct effect of cold on the forearm vessels, or an axon reflex in the somatic nerve supply. Considering the penetrative effect of cold, the latter theory appears to be more credible.,

The results of such blood flow studies, cast doubts on the hypothetical bases for the therapeutic use of cold. While the peripheral changes that take place in response to local cooling are reasonably well documented, the deeper internal circulatory changes remain vague, and are the subject of much speculation. At times deep vascular changes may parallel or complement peripheral changes, but on other occaisions, they may be compensatory for homeostatic purposes (Murphy, 1960, Keatinge & Harman, 1980).

Contraindications

The therapeutic effects of cold therapy are unquestionable, but in rare situations, it may have deleterious effects. Grant (1964), discovered no contraindications in 7000 outpatients which he treated with ice. He did however, avoid symptomatic rheumatoid arthritis patients, as such conditions could respond with increased pain and joint stiffness (Barnes, 1979). Any body areas with compromised circulation (for example some diabetic conditions and Raynauds syndrome), could become more ischemic with cooling (Kramer, 1977, Barnes, 1979). Short exposures of extreme cold have been shown to increase cardiac contractile force, and raise the arterial blood pressure (Murphy, 1960, Waylonis, 1967). Haines (1967), avoids cold over the heart or left shoulder areas, although Laing et al

(1973), suggest that it is the anxiety associated with this, rather than the cardiac effects, which is the most threatening.

Persons who have sensory impairment, are paralysed or comatose, may be at risk of suffering a cold injury, if they are unable to distinguish the sensations associated with cold (Kramer, 1977, Barnes, 1979).

A hypersensitivity to cold has been noted in a few patients. This reaction is probably related to a "histamine-like" release in the skin, causing hive-like eruptions (Olson & Stravino, 1972).

The above mentioned conditions are relatively rare in athletic populations, where the main hazards of cold treatment are limited to ice burns, and edema caused by prolonged usage (Matsen et al, 1975, McMaster & Liddle, 1980). Ice therapy can be an exceptionally safe form of treatment, provided that attention is paid to tissue viability.

III. METHODS AND PROCEDURES

A. Subjects

The present study examined the right forearm blood flow of 20 university student volunteers. The age of the volunteers ranged from 20 to 33 yrs., with a mean age of 25.5 ± 3.35 yrs. All subjects were screened for cardiovascular anomalies prior to testing. The use of the right arm only for blood flow measurements, was necessitated by the set up of the experimental apparatus.

The subjects were assigned to two groups. Group A consisted of 10 subjects who experienced a 5 ℃ water bath first, and group B of 10 subjects who were immersed in a 10 ℃ water bath first. All subjects experienced both of the experimental temperatures, but the order of presentation was reversed.

Of the 20 subjects, one was screened out prior to testing, because of an adverse reaction of nausea, dizziness and visible distress, in response to occlusion cuff inflation. Data for another three subjects were removed prior to analysis, because of incomplete data.

B. Environment

All of the blood flow measurements were conducted in a thermostatically controlled laboratory, with the room temperature ranging from 23.5 to 26 ℃. The mean room temperature throughout the study was 24.7 ± 0.7 ℃. During the experiment all of the subjects were clothed in normal street attire, but all wore a light cotton t-shirt with loose fitting sleeves on their upper bodies. Blood flow measurements were carried out during the months of October, November and December, with the ambient temperatures ranging from -11 to +18 ℃. The average high temperature over this period was 2.03 ℃, and the average low -5.43 ℃.

C. Instrumentation

Forearm anthropometric measurements were made using a Gulick anthropometric tape. [1]

Blood flow estimation was made using double stranded mercury in silastic strain gauges. [2] The strain gauges were attached to the forearm using perspex mounts, similiar to the Whitney mount (Whitney, 1953). These perspex mounts (plate 1) differ from the Whitney mount, in that the support pillars were contoured to fit the shape of the forearm, and allow better gauge compliance. The mounting system used by Whitney, was reversed for the present study, so that instead of a slight gap between the ends of the strain gauge, a slight overlap occurred. The strain gauges were selected to suit the arm circumference of the subject, so that adequate adjustment was available within the perspex mount, to shorten or lengthen the gauge, reproducing a 20 g tension.

Plate 1

Perspex strain gauge mount

[1]J.A.Preston corporation, 71 Fifth Ave., New York, N.Y. 10003
[2]Parks Medical Electronics Inc., Beaverton, Oregon 97075

For recordings, the strain gauge was connected as one arm of a bridge circuit, the output of which, was recorded on a polygraph. (Beckman Offner Type RB Dynograph Recorder with a 9875 coupler)[3]

No temperature compensation was used for the present study. The effect of temperature on strain gauge sensitivity, was checked using a water filled bladder, and was not found to be significant. (Appendix C)

Venous outflow from the forearm was occluded using a proximal occlusion cuff 9 cm. in width, fastened around the upper arm. This cuff was connected to a 5 gallon constant pressure drum, by wide bore plastic tubing (1.5 cm I.D.), allowing rapid cuff inflation, using a solenoid valve, to 65 mm Hg, during recording. As each subject acted as their own controls, a standard cuff occlusion pressure of 65 mm Hg was used, and provided suitably reproducible blood flow traces in all experiments. The venous occlusion cuff was deflated via a solenoid valve opening to negative pressure. immediately after 5 sec. of occlusion. A distal wrist cuff, 4 cm. in width, was made from a bicycle inner tube, and was placed around the wrist to occlude hand blood flow. This cuff was inflated to 250 mm Hg from a constant pressure laboratory source, and was deflated passively.

A perspex water bath[4] (Plate 1) was used to maintain the subject's forearm in a standardised experimental position throughout the monitoring period. This bath allowed the subject's hand to be sealed outside of the water bath, by means of a neoprene sleeve and the wrist occlusion cuff. Water was introduced into the bath without disturbing the experimental position of the subject's forearm. Filling of the water bath was by siphon, assisted by an electrical pump.

Water at the experimental temperature was obtained from a 564 l. water tank, in which the water was constantly stirred, and the temperature was maintained at a constant temperature ± 0.2 °C, by a refrigeration unit.

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[3] Offner Division of Beckman Instruments Inc., 3900 River Rd., Schiller Park, Illinois

[4] 35.5 cm x 16 cm x 16.5 cm, capacity 9.09 l.

Water tank temperature, experimental bath temperature, and the forearm skin temperature were monitored with copper/constantan thermocouples read with an analog meter (Bat-4 laboratory thermometer).[5]

Plate 2

Perspex water bath with neoprene sleeve

D. Testing Procedure

Anthropometric Measurement

The subject's right arm was used for all blood flow measurements. The distance between the right lateral humeral epicondyle and the right ulnar styloid process was measured to

[5] Bailey Instruments Inc., Saddlebrook, N.J. 07662

the nearest millimeter, with the anthropometric tape, with the subject's arm in the experimental position. This experimental position had the subject's elbow joint flexed to 90 ° and the forearm fully pronated. Water fast marks were made on the extensor surface of the forearm, at 25%, 50%, and 75% of the length. The mark at 25% of the length from the lateral epicondyle, was the proximal monitoring site, and the 75% mark was the distal monitoring site. The midpoint of the forearm was designated by the 50% mark. These marks were retained by the subject's throughout the experiment, although five remeasurements were required over the 40 trials. The forearm girth was measured at the proximal and distal monitoring points, with these circumferences recorded as the baseline girths. These girths were remeasured at the beginning of each trial, before the strain gauges were positioned.

Gauge Attachment

The mercury in silastic strain gauge was clamped into the perspex arm mounts, and these were attached to the extensor surface over the forearm, using micropore surgical tape Care was taken to position the mounts so that their midlines were directly over the proximal and distal marks. The strain gauge encircled the arm as a double strand, with the free end being hooked over the attachment reel (plate 1). The gauges were also held in place by 2 cm. lengths of clear plastic tygon tubing [6] (2 cm. I.D.),split lengthwise. Three of these were positioned around the circumference of the arm with micropore tape, and the arch of these devices, prevented the gauges from rolling proximally or distally from the target cross section, without interfering with the gauge function.

Experimental Positioning

With the gauges in position, the subject's arm was then located in the water bath, so that the hand protruded through the neoprene sleeve. The distal wrist cuff was applied around the wrist, as per Kerslake (1949), at the level of the styloid processes, and this was secured in

[6] Fisher Scientific Co., 711 Forbes Ave., Pittsburgh, Pensylvania 15219.

place with a locking strap. The subject's arm was then pulled further back into the tank, until the wrist cuff was flush with the perspex tank end.

Seat height was adjusted, so that the subject's arm was flexed to 90 °, the forearm was fully pronated, and neither the arm nor the strain gauge mounts, contacted any part of the tank. Although most investigators suggest that the measured limb should at least be at heart level (Cooper et al, 1955, Eagan, 1961), a dependent position was adopted for the present study. This position was used because it replicated the positions used during cold therapy, and provided reproducible blood flow recordings. It also allowed the hand to be left free of the water bath throughout the experiment, in this way avoiding the pain often associated with cold immersions.

The elbow pad was adjusted, so that the experimental position was maintained, and the strain gauges were kept free from the bottom of the tank. A pad was also placed under the hand so that the wrist was slightly flexed, and in a comfortable position which required no muscular effort to maintain.

Plate 3

Experimental position for blood flow recording

Occlusion Cuffs

The proximal venous occlusion cuff was placed as high up the arm as possible, with the upper edge against the axilla. This was necessary to keep the cuff free of the water bath, and any subsequent movement artifacts caused by bouyancy changes. It was ensured that the upper arm was always free of the sides of the tank, preventing any compression of the vasculature, or any movement artifact due to cuff inflation.

Temperature Monitoring

One of the copper/constantan thermocouples was taped to the extensor surface of the forearm at the midpoint, using micropore tape.

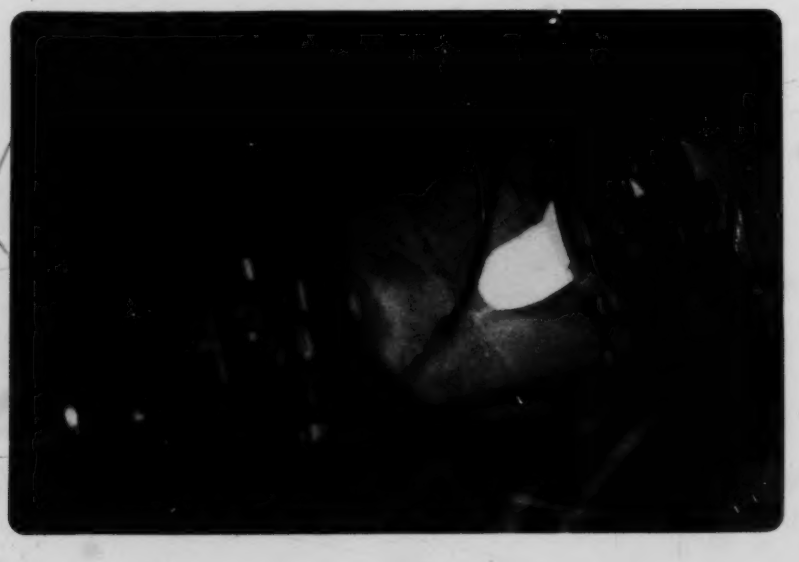

Plate 4

Strain gauge and thermocouple attachment

The other thermocouple was taped to the perspex wall of the tank, 2 cm. away from the side and 2 cm. from the water inlet to provide measurements of water temperature.

Gauge Tension Setting

The Offner recorder was switched on at least 30 min. prior to each test to allow adequate warm up before experimentation. The dynograph amplifiers were preset to a sensitivity of 50 μV/cm, and the bridge balance control adjusted to a resistance appropriate for the strain gauges being used (equivalent to a 20 g tension). Using the tension adjusting nuts, the strain gauges were tightened around the forearm to produce a tension equivalent to a 20 g weight suspended from the gauge. When the bridges were balanced and each gauge was adjusted to a 20 g tension, the subject was left to rest for 5 min.

Blood Flow Recordings

The distal arterial occlusion cuff was inflated to 250 mm Hg from a constant pressure source. After 1 min. of wrist occlusion, the first blood flow recordings were made at t = 0. On triggering, the proximal occlusion cuff automatically inflated to 65 mm Hg, preventing venous outflow, but permitting arterial inflow. Simultaneously the chart paper speed increased from 10 mm/min. to 10 mm/sec., and proximal and distal plethysmographic recordings were made. After 5 sec., the proximal cuff was deflated, and the paper speed again slowed to 10 mm/min. The second recording was made at t = 55 sec., and subsequently every 60 sec. thereafter. The wrist cuff was released for 45 sec. out of every 5 min., to maintain nutritive blood flow, and for subject comfort. That is, the cuff was released from t = 4 min. to t = 4 min. 45 sec., again at 9 min., and so on.

At t = 10 min., the water bath was filled with water at the experimental temperature. A small electrical pump assisted the filling, giving an average filling time of 28 $\pm$ 0.7 sec.

Blood flow was recorded with the experimental temperature water in the tank, for 30 min., and at t = 40 min., the water was drained from the tank. This drainage was by gravity

siphon only, and required an average of 43 ± 0.9 sec. for complete emptying. A further 30 min. of blood flow recordings were made, and at t = 70 min., the experiment was terminated.

Forearm temperature from the thermocouple at the midposition of the forearm, was noted every min. for the 70 min. of testing.

E. Data Analysis

Blood flow was calculated from each of the plethysmographic inflow traces recorded. The calculations were made as described in Appendix B.

Because of incomplete data, three subjects were discarded from the study. Because of the high variability in resting blood flow values observed, the changes in blood flow were calculated for each subject under each condition. Abramson, Zazeela and Marrus (1939), noted a marked variation in the absolute resting blood flow values of apparently normal subjects. Differences were observed for the same individuals measured on different days. Hyman, Burnap and Figar (1963), found significant bilateral differences in forearm blood flow at rest. Lind and McNicol (1968), measured forearm blood flow following muscular exercise. Although the levels of individual blood flow varied, each subject demonstrated the same pattern of response.

The reasons for such variability have not been successfully defined. Postulated causes include; normal variations in cardiac output (Abramson et al, 1939), autonomically induced variations in skin blood flow (Hyman et al, 1963), environmental stimuli (Sparks, 1978), and emotional reactions (Roddie, 1983).

Humphreys and Lind (1963), allowed for these individual variations in blood flow, by calculating the increase in blood flow over the mean resting flow. As the focus of the present study is the changes in forearm blood flow with cooling, a similiar treatment of raw data has been applied. For each subject the mean resting blood flow was calculated for each monitoring site (proximal and distal), on each occasion. Relative blood flow was derived, by

subtracting the mean resting flow from each of the thirty (30) recordings made during cold immersion, and the thirty (30) subsequent recordings. Mean blood flow deviations were calculated for every 3 min. to provide 10 experimental data points and 10 post-experimental points. The experimental data points, consisted of the mean flow deviations for minutes 12,13,14 / 15,16,17 / 18,19,20 / 21,22,23 / 24,25,26 / 27,28,29 / 30,31,32 / 33,34,35 / 36,37,38 / 39,40. And the post-experimental data points are the means of minutes 42,43,44 / 45,46,47 / 48,49,50 / 51,52,53 / 54,55,56 / 57,58,59 / 60,61,62 / 63,64,65 / 66,67,68 / 69,70.

These transformations resulted in one (1) mean control blood flow value, ten (10) experimental, and ten (10) post-experimental values.

Using this transformed data, immersion temperature and immersion time were examined with six (6) two way analyses of variance. Repeated measures were made over the immersion time and immersion temperature factors, with proximal and distal blood flow being analysed separately. For each of the blood flow monitoring sites, three (3) two way analyses of variance were made. The first examined experimental blood flow deviations, the second contrasted post-experimental deviations in flow, and the third analysis of variance examined experimental and post-experimental deviations together.

Mean forearm temperatures for each minute of cold immersion and recovery, were calculated.

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IV. RESULTS

A. Forearm blood flow

The major purpose of the present study, was to determine the effects of two different immersion temperatures, on the cutaneous and skeletal muscle blood flow to the forearm.

Nineteen (19) healthy, informed, male volunteers, with no reported cardiovascular anomalies, acted as subjects, with only the subject's right arm being used for measurements. The age and anthropometric statistics of all subjects are included in Table 1.

The results and discussion are based on the effects of two therapeutic immersion temperatures (5 °C and 10 °C), on proximal and distal blood flow, during 30 min. of cooling (experimental), and 30 min. subsequent to the cooling (post-experimental). The distal and proximal blood flow deviations and standard deviations of the transformed data, are presented in Tables 2 and 3 respectively. The average resting blood flows were 1.71 ml/100ml of tissue/min. and 1.88 ml/100 ml of tissue/min. for the distal and proximal portions respectively. Graphic representation of the distal and proximal blood flow alterations, are shown in Figures 1 and 2 respectively.

The two way analysis of variance (ANOVA) tests with repeated measures on both the immersion temperature factor, and the immersion time factor, for the distal blood flow during the experimental period, provided no significant main effects for time or temperature(Table 4). This ANOVA did however, produce a significant interaction effect ($p<0.05$)

The analysis for the post-experimental distal blood flow deviations (Table 5), demonstrated a significant main effect for time ($p<0.001$), and a significant interaction between time and immersion temperature ($p<0.001$). The temperature main effect was not significant.

With the experimental and post-experimental distal blood flow changes analysed together (Table 6), no significant temperature effect was observed.

Table 1

Subject statistics

CHARACTERISTIC	RANGE	MEAN	S.D
AGE (yrs)	20 - 33	25.55	3.35
ARM LENGTH (cm)	24 - 30	27.03	1.29
CIRCUMFERENCE PROXIMALLY (cm)	26.4 - 30	27.90	.96
CIRCUMFERENCE DISTALLY (cm)	17.9 - 21.6	18.85	.96

Table 2
Mean distal blood flow deviations
average of three points (ml./100 ml. of tissue/min.)

	5 °CELSIUS		10 °CELSIUS	
TIME	MEAN	STD. DEVN.	MEAN	STD. DEVN.
1	-0.751	0.680	-0.606	0.696
2	-0.764	0.561	-0.760	0.534
3	-0.722	0.624	-0.806	0.491
4	-0.721	0.639	-0.894	0.524
5	-0.755	0.523	-0.912	0.608
6	-0.721	0.646	-0.882	0.717
7	-0.697	0.783	-0.868	0.567
8	-0.635	0.715	-0.839	0.511
9	-0.616	0.780	-0.738	0.581
10	-0.546	0.680	-0.774	0.571
11	-0.129	0.611	-0.817	0.553
12	-0.506	0.701	-0.905	0.590
13	-0.745	0.686	-1.005	0.563
14	-0.892	0.708	-0.971	0.526
15	-0.890	0.712	-0.947	0.575
16	-0.942	0.628	-1.032	0.582
17	-0.965	0.667	-1.044	0.600
18	-0.897	0.613	-1.042	0.631
19	-0.969	0.568	-1.052	0.666
20	-0.876	0.628	-1.052	0.524

Table 3
Mean proximal blood flow deviations
average of three points (ml./100 ml. of tissue/min.)

	5 °CELSIUS		10 °CELSIUS	
TIME	MEAN	STD. DEVN.	MEAN	STD. DEVN.
1	-0.273	0.447	-0.312	0.449
2	-0.402	0.410	-0.441	0.295
3	-0.427	0.520	-0.562	0.264
4	-0.421	0.353	-0.656	0.261
5	-0.357	0.310	-0.626	0.366
6	-0.327	0.513	-0.552	0.352
7	-0.250	0.554	-0.573	0.518
8	-0.216	0.416	-0.598	0.321
9	-0.216	0.484	-0.601	0.432
10	-0.176	0.526	-0.544	0.463
11	+0.040	0.587	-0.602	0.457
12	-0.405	0.656	-0.683	0.441
13	-0.613	0.500	-0.802	0.412
14	-0.778	0.390	-0.866	0.411
15	-0.777	0.489	-0.745	0.563
16	-0.733	0.616	-0.876	0.572
17	-0.904	0.478	-0.832	0.514
18	-0.867	0.432	-0.699	0.572
19	-0.927	0.389	-0.759	0.637
20	-0.884	0.421	-0.846	0.559

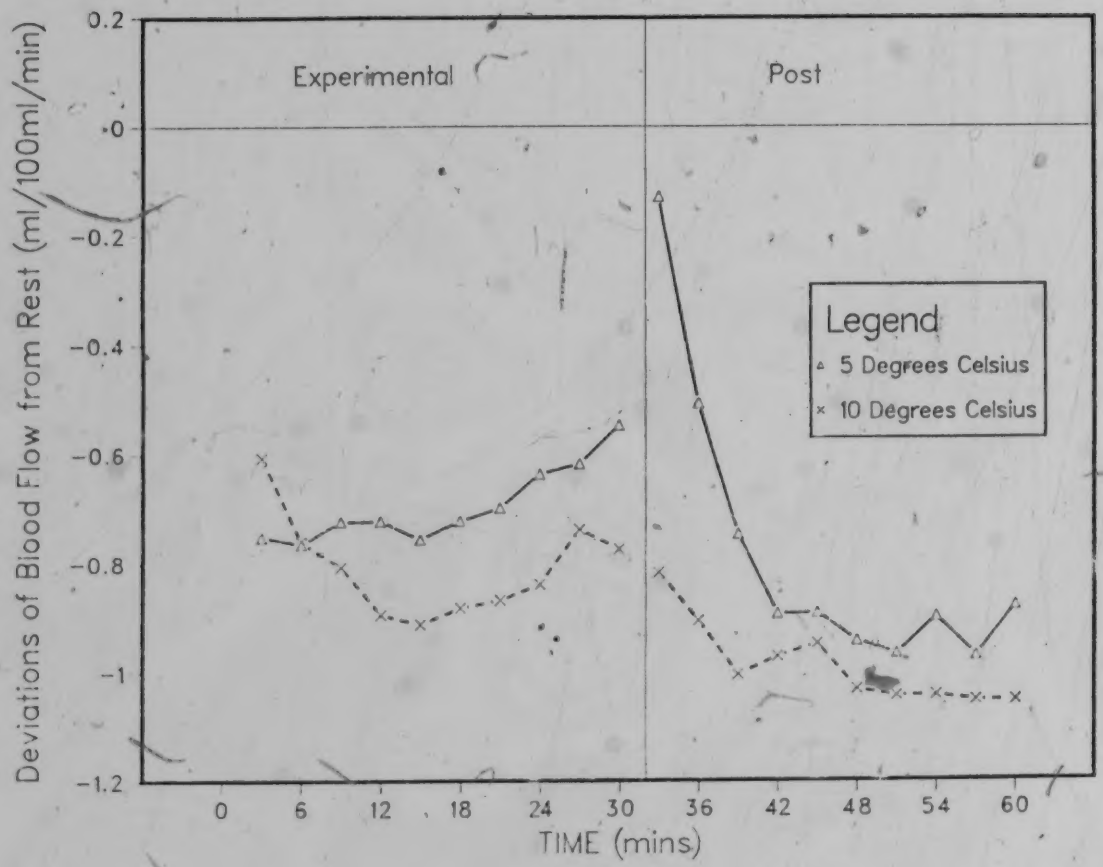

Figure 1. Distal blood flow deviations

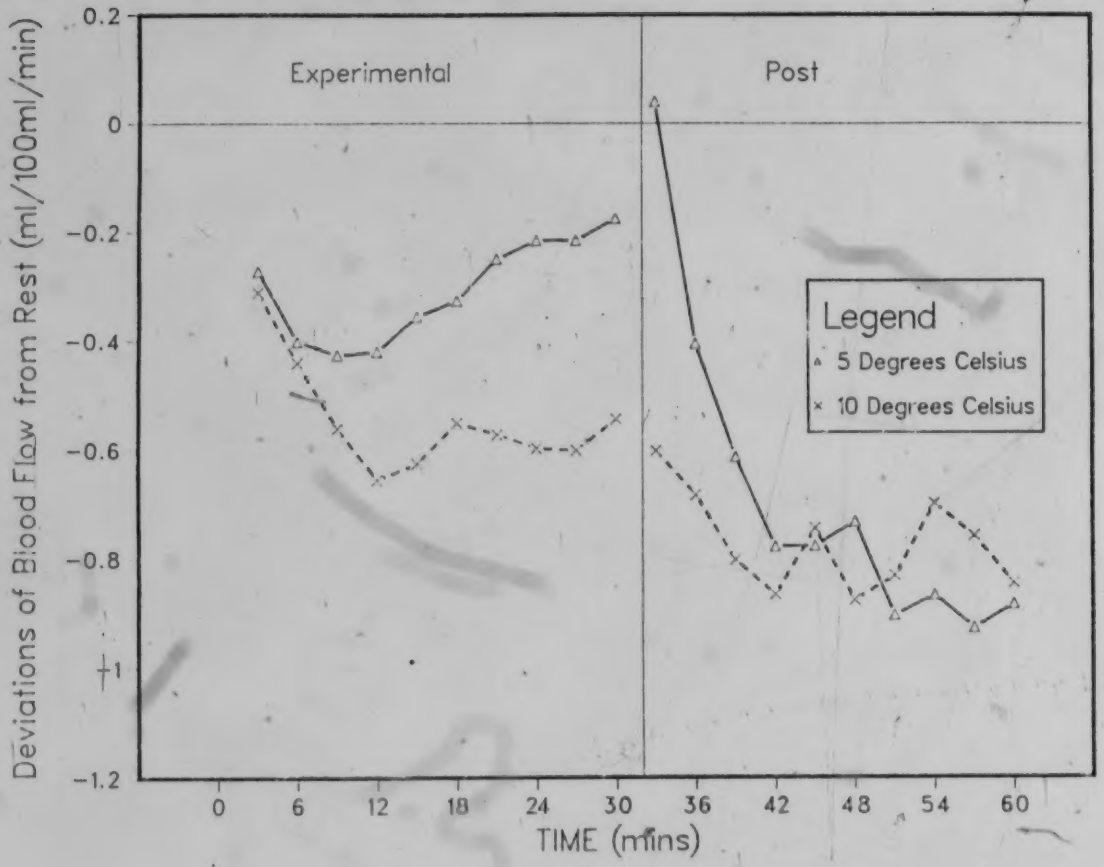

Figure 2. Proximal blood flow deviations

Again significant time effects ($p<0.001$), and interactions ($p<0.001$) were demonstrated.

A two way ANOVA of proximal blood flow changes during the experimental period (Table 7), revealed a significant main temperature effect ($p<0.05$), with no significant interaction or time effects. During the post-experimental phase (Table 8), the time factor had a significant effect on proximal blood flow ($p<0.001$), and a significant interaction ($p<0.001$) effect. The temperature factor did not have a significant effect.

The final two way analysis of variance, combining proximal blood flow deviations during the experimental and post-experimental periods (Table 9), produced no significant temperature effect, but a significant immersion time effect ($p<0.001$), and a significant interaction ($p<0.001$).

The results of these analysis of variance tests are presented in tables 4 to 9.

B. Forearm temperature

The mean forearm temperatures, and standard deviations during the experimental and post-experimental periods, for the 5 °C and 10 °C treatments, are presented in Tables 10 and 11 respectively. The graphic representation of these data points is shown in Figure 3.

Table 4

Analysis of variance for distal blood flow

deviations - experimental

SOURCE OF VARIATION	SUM OF SQUARES	DEGREES FREEDOM	MEAN SQUARES	F RATIO
Temperature main effects	1.063	1	1.063	0.743 N.S.
Temperature X subjects within groups	21.445	15	1.430	
Time main effects	1.058	9	0.118	1.843 N.S.
Time X subjects within groups	8.615	135	0.064	
Temperature X time interaction	0.908	9	0.101	1.956 *
Temperature X time X subjects within groups	6.968	135	0.052	

* $p<0.050$

Table 5

Analysis of variance for distal blood flow deviations - post-experimental

SOURCE OF VARIATION	SUM OF SQUARES	DEGREES FREEDOM	MEAN SQUARES	F RATIO
Temperature main effects	3.387	1	3.387	2.063 N.S.
Temperature X subjects within groups	24.622	15	1.641	
Time main effects	8.293	9	0.921	14.293 **
Time X subjects within groups	8.704	135	0.035	
Temperature X time interaction	2.884	9	0.320	9.036 **
Temperature X time X subjects within groups	4.787	135	0.035	

** $p<0.001$

Table 6
Analysis of variance for distal blood flow deviations - experimental & post-experimental

SOURCE OF VARIATION	SUM OF SQUARES	DEGREES FREEDOM	MEAN SQUARES	F RATIO
Temperature main effects	4.122	1	4.122	1.506 N.S.
Temperature X subjects within groups	41.047	15	2.736	
Time main effects	12.204	19	0.642	7.049 **
Time X subjects within groups	25.968	285	0.091	
Temperature X time interaction	4.120	19	0.217	3.684 **
Temperature X time X subjects within groups	16.775	285	0.059	

Table 7
Analysis of variance for proximal blood flow deviations - experimental

SOURCE OF VARIATION	SUM OF SQUARES	DEGREES FREEDOM	MEAN SQUARES	F RATIO
Temperature main effects	4.613	1	4.613	6.449 **
Temperature X subjects within groups	10.729	15	0.715	
Time main effects	1.438	9	0.160	1.626 N.S.
Time X subjects within groups	13.263	135	0.098	
Temperature X time interaction	1.261	9	0.140	1.578 N.S.
Temperature X time X subjects within groups	11.986	135	0.089	

Table 8
Analysis of variance for proximal blood flow deviations - post-experimental

SOURCE OF VARIATION	SUM OF SQUARES	DEGREES FREEDOM	MEAN SQUARES	F RATIO
Temperature main effects	0.595	1	0.595	0.496 N.S.
Temperature X subjects within groups	17.999	15	1.200	
Time main effects	9.751	9	1.083	11.543 **
Time X subjects within groups	12.672	135	0.094	
Temperature X time interaction	4.341	9	0.482	5.722 **
Temperature X time X subjects within groups	11.380	135	0.084	

Table 9
Analysis of variance for proximal blood flow deviations - experimental & post-experimental

SOURCE OF VARIATION	SUM OF SQUARES	DEGREES FREEDOM	MEAN SQUARES	F RATIO
Temperature main effects	4.261	1	4.261	2.554 N.S.
Temperature X subjects within groups	25.028	15	1.669	
Time main effects	25.733	19	1.354	9.613 **
Time X subjects within groups	40.153	285	0.141	
Temperature X time interaction	6.549	19	0.345	3.630 **
Temperature X time X subjects within groups	27.067	285	0.095	

TABLE 10

Average forearm temperatures during cooling
5 °C water bath

	COOLING		RECOVERY	
TIME	MEAN	S.D.	MEAN	S.D.
Rest	31.39	1.09		
1	17.78	2.35	9.99	.97
2	14.51	1.75	11.87	1.01
3	13.45	1.80	13.16	1.27
4	12.43	1.67	14.14	1.19
5	11.75	1.48	14.80	1.24
6	11.27	1.49	15.71	1.28
7	10.82	1.37	16.29	1.18
8	10.41	1.30	16.72	1.23
9	10.13	1.39	17.16	1.20
10	9.88	1.33	17.40	1.16
11	9.74	1.35	17.71	1.12
12	9.51	1.35	17.95	1.14
13	9.43	1.37	18.24	1.06
14	9.23	1.37	18.48	1.06
15	9.16	1.39	18.65	.99
16	9.05	1.40	18.91	1.06
17	8.87	1.29	19.01	1.12
18	8.76	1.17	19.16	1.02
19	8.65	1.16	19.30	1.05
20	8.61	1.06	19.46	.96
21	8.58	.99	19.59	.99
22	8.50	.96	19.68	.98
23	8.33	.93	19.81	.91
24	8.32	.89	19.90	.98
25	8.28	.91	20.02	.95
26	8.20	.86	20.21	.94
27	8.16	.84	20.24	.91
28	8.13	.88	20.34	.89
29	8.06	.83	20.41	.88
30	7.96	.78	20.44	.89

TABLE 11

Average forearm temperatures during cooling
10 °C water bath

	COOLING		RECOVERY	
TIME	MEAN	S.D.	MEAN	S.D.
Rest	31.42	.87		
1	19.76	1.99	13.45	.87
2	17.92	1.53	15.03	.65
3	16.83	1.30	15.92	.57
4	16.10	1.33	16.52	.61
5	15.59	1.23	17.20	.71
6	15.17	1.20	17.80	.70
7	14.77	1.20	18.20	.90
8	14.44	1.10	18.42	.84
9	14.12	.96	18.64	.81
10	13.77	.90	18.92	.80
11	13.69	.92	19.21	.77
12	13.47	.79	19.41	.86
13	12.62	.80	19.53	.86
14	13.18	.65	19.76	1.01
15	12.99	.64	19.84	.99
16	12.89	.60	20.09	.99
17	12.87	.72	20.16	.99
18	12.81	.72	20.31	.92
19	12.70	.63	20.38	.93
20	12.55	.53	20.42	.84
21	12.48	.52	20.74	1.10
22	12.43	.55	20.77	1.08
23	12.46	.69	20.82	1.06
24	12.36	.62	20.91	1.02
25	12.29	.64	20.95	.96
26	12.18	.47	21.07	1.03
27	12.23	.44	21.17	1.11
28	12.17	.47	21.26	1.06
29	12.10	.52	21.20	1.01
30	12.07	.54	21.16	.84

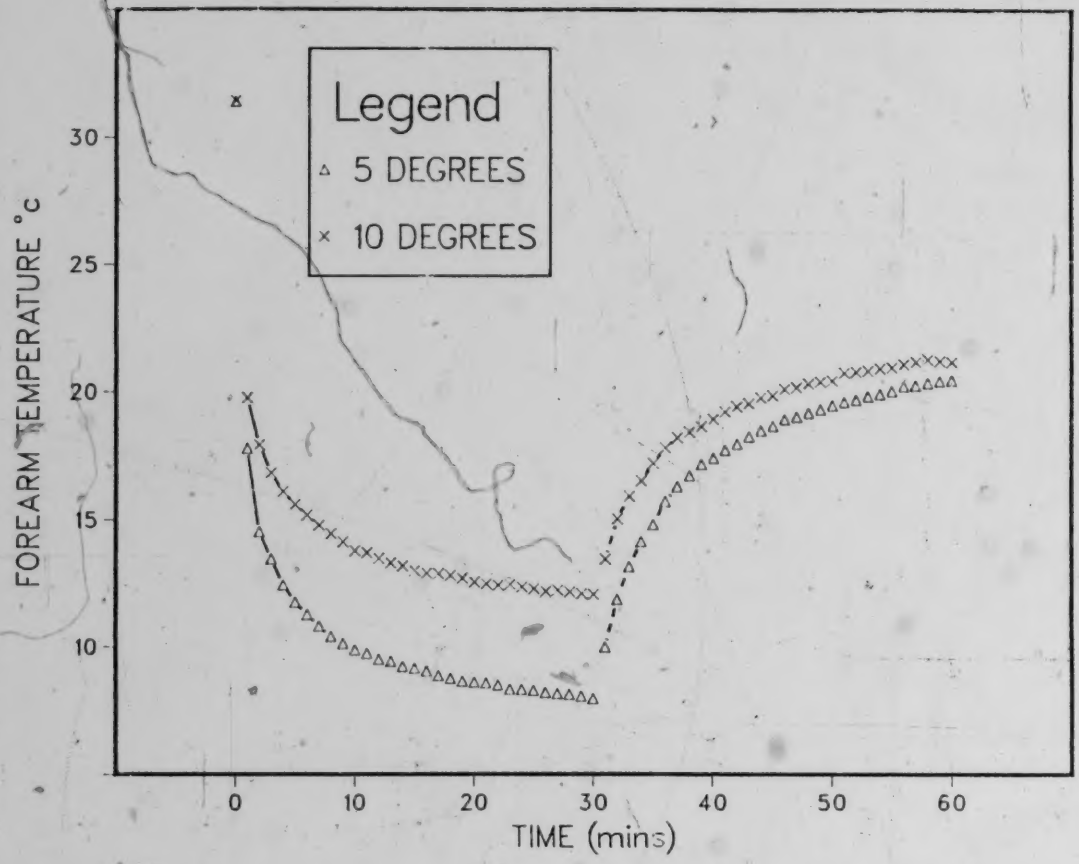

Figure 3. Forearm skin temperature with cooling

V. DISCUSSION

A. Forearm Blood Flow

The analysis of variance tests conducted for proximal and distal blood flow during the treatment periods, demonstrated a variety of statistically significant effects for immersion time, immersion temperature, and interactions. (Tables 4 to 9).

As described above, the distal blood flow measurement is considered to include a high skin blood flow component, with very little contribution from skeletal muscle flow (Cooper et al, 1955, Clarke et al, 1958). For proximal blood flow measurement, the reverse holds true, with the high muscle to skin ratio at this point, resulting in an estimated flow, indicative of skeletal muscle flow. For the purposes of the present study, the exact contributions of skin and muscle circulation to the blood flow measurements, could not be discerned. The discussion of results has been based on the assumption, that this technique, which has been validated elsewhere (Clarke et al, 1958), indicates cutaneous and skeletal muscle circulatory changes.

It is evident from tables 4, 5 and 6, that the immersion temperature had no significant effect on distal blood flow changes. To this investigators knowledge, no previous studies have examined the effect of immmersion temperature on distal (or cutaneous) blood flow. From a physiological perspective, it would appear that the two immersion temperatures had similiar influences on cutaneous flow, through a direct effect on the smooth muscle cells of the cutaneous vessels, and stimulation of the cutaneous cold receptors.

In the present study, distal blood flow was not found to significantly decrease during the experimental period, but decreased significantly during the post-experimental period, and when the experimental and post-experimental period were examined together as one treatment. Clarke, Hellon and Lind (1958), have reported distal blood flow changes for 1 °C forearm immersion. Their data suggested a slight increase in distal blood flow with cooling, but this was not statistically treated. Knight and Londeree (1980), measured ankle blood flow during

-7 °C cold pack cooling. Blood flow was measured at a point 5 cm above the (lateral?) malleolus, a measurement which presumably includes a high skin, tendon, bone component, with very little underlying muscle. Blood flow during the 45 min. treatment period (25 min. cooling and 20 min. of post-cooling), was found to be significantly lower than control blood flow.

Most researchers have inferred blood flow changes from cutaneous temperature alterations (Lewis, 1930, Knight et al, 1980), or from plethysmographic data obtained from acral body parts (Greenfield et al, 1951, 1952). These findings suggest that a vasoconstriction will occur initially, followed by a vasodilation or "hunting reaction". From figure 1, it appears that distal blood flow in the present study, was reduced with cooling, the largest reduction in flow occurring after approximately 7 min. of 5 °C cooling. At this point, blood flow is reduced 47.4% from the average pre-immersion blood flow, and the average forearm skin temperature was between 10.8 and 11.8 °C. For the 10 °C experimental period, a reduction of 50.39% in blood flow occurred at 14-16 min. and the average forearm temperature was between 12.9 and 13.2 °C. Keatinge and Harman (1980), suggest that if the tissue temperature falls below 12 °C, a reflex vasodilation is likely. No reflex vasodilations or hunting reactions were observed in the present study. This may have been because the treatment temperatures used were not sufficiently cold to penetrate and lower the tissue temperatures to below 12 °C, or because of the lack of reactivity in the forearm cutaneous circulation. On the other hand, this might be attributed to the lack of arterio-venous anastamoses in the forearm skin (Roddie, 1983). These structures are considered essential for cold induced vasodilations or hunting reactions to occur (Kalenak et al, 1975).

The finding that the distal blood flow continued to decrease for 30 min. after cooling, does not correspond with Greenfield's (1963) conclusions, based on a review of the after effects of cold. His review was based on the temperature changes of the skin of acral body parts, and suggests that following the removal of a cooling source, a vasodilation occurs, and persists to a point where the blood flow may surpass that of the untreated contralateral limb.

The finding in the present study concurs with the result of Knight and Londeree's (1980) study, which noted a reduced ankle blood flow for 20 min. after cold treatment. As described above, much of the cold induced vasodilation, hunting reaction and after effect vasodilation, have been attributed to the involvement of arterio-venous anatamoses. With the forearm being devoid of these highly reactive structures, such reactions are not often seen (Roddie, 1983). A significant interaction between the immersion temperature and immersion time, is demonstrated for all of the analyses of distal blood flow. The interaction during the experimental period is not particularly strong ($p<0.049$),and is likely due to variability within the blood flow. For the post experimental period, the blood flow curves appear to converge with time, as the forearm gradually rewarms.

Most theories on cold therapy, suggest that cold modalities should be removed prior to a cold induced vasodilation occurring. This time is estimated as 10 min. by Wise (1973), 5 to 12 min. by Hocutt (1981), and 20 min. by Moore et al (1966). Data and statistical analysis from the present study would suggest that forearm distal blood flow is significantly reduced for up to 30 min. following a 30 min. immersion in 5 °C or 10 °C water. No evidence for a reactive vasodilation in distal blood flow, was provided for these temperature treatments.

Of note however, is the sharp increase in blood flow, both proximally and distally, after the 5 °C water was removed from the forearm (Figures 2 and 3). This effect is dramatic and only appears to persist for 5-7 min. after cold removal. The effect could be described as either the result of a hydrostatic pressure removal, or a reactive hyperemia. The concept of hydrostatic effects on plethysmographic blood flow estimation, has been discussed above. The influence of hydrostatic pressure on forearm blood flow measurement is considered to be minimal (Dahn and Hallböök, 1970, Francis and McCaig, 1985), and as in the present study, the water level was only between 2 and 3 cm above the extensor surface of the forearm, hydrostatic pressure was not expected to be a factor. If the hydrostatic effect was significant, a similiar effect for both the 5 °C and 10 °C treatments would be expected. A reactive hyperemia appears to be a more plausible explanation for the observed blood flow increases.

A visible hyperemia is evident in plate 5, which demonstrates a distinct reddening of the skin with piloerection. This reaction was observed in all parts of the forearm which were immersed in the 5 °C water.

Plate 5

Reactive hyperemia of the forearm following

a 30 min. 5 °C immersion

The mechanisms involved in reactive hyperemia are not clear according to Sparks (1978), and probably involve a metabolic component related to hypoxia of the local vasculature and tissues. Greenfield (1963), proposes that "chemical substances" accumulate during circulatory interruption, and promote a vasodilation when circulation is restored. Another theory suggests that the reduced transmural pressure in the vasculature during restricted circulation, initiates a myogenic response of local dilation (Greenfield, 1963, Sparks, 1978). All of these mechanisms may be operative, but the key to the reaction, is the fact that the forearm treated in 10 °C

water, did not display the reaction. As distal flow is not significantly different for the two temperature treatments , metabolic debts or reduced transmural pressure, can not account for the hyperemia. Presumably, the 10 °C immersion, does not cool the skin sufficiently to initiate the production of vasodilatory substances subcutaneously. The reactive hyperemia demonstrated for the proximal measurement, is not as intense as the distal effect, and during the cooling phase the proximal blood flow was higher under the 5 °C treatment, than for the 10 °C. This suggests that a reactive hyperemia of the skeletal muscle component of flow was unlikely, and the increased flow observed, was probably the expression of the cutaneous portion of the proximal blood flow.

The analysis of variance results for the proximal blood flow (tables 7 to 9), indicate that the immersion temperature only had a significant effect on blood flow during the cooling period. This result is in accord with the findings of Clarke, Hellon and Lind (1958), who found that blood flow to the forearm was relatively unchanged during 10 °C cooling, increased slightly at 6 °C, and at 1 °C, the increase was dramatic, although unsupported by statistical analysis. Through the use of proximal and distal strain gauges, and adrenaline iontophoresis, it was established that this increased flow was predominantly to the skeletal muscle. The differences in proximal blood flow with immersion temperature is illustrated in Figure 3. The direction of the change, in the present study, differs from the results obtained by Clarke et al (1958). While these workers found that the proximal blood flow increased with decreasing immersion temperatures, the present study indicates that both immersion temperatures reduced the proximal blood flow, with the 10 °C temperature being more effective. With the range of experimental temperatures employed for the present study, it is unlikely that the penetration of cold into the deeper tissues, was sufficient to cause changes in the vascular smooth muscle (Bierman, 1939), and mediate a change in the blood flow. According to Clarke et al (1958), changes to the skeletal muscle blood flow are the result of an axon reflex in the somatic nerve supply. Preliminary work by these authors, revealed that forearm circulation decreased with 42 °C to 14 °C immersion, increased slightly at 10 °C, and underwent a marked

increase at 2 °C. The mechanisms for these changes are unclear, but it is suggested by the investigator, that at an optimum immersion temperature, the muscle blood flow and cutaneous blood flow are complementary, working to maintain the thermal gradient. At temperatures below this, the blood flows become countereffective, with cutaneous blood flow reduced to prevent the cooling, and the muscle blood flow increased to maintain tissue viability.

The analyses of variance did not demonstrate a significant change in proximal blood flow during the cooling period, but significant changes were noted during the post-experimental period, and when the experimental and post-experimental periods were examined together as a treatment. Proximal blood flow was at it's least at 8-10 min. for the 5 °C treatment, where it was 22.14% below pre-immersion levels. For the 10 °C treatment, the lowest average flow recorded was at the 11-13 min. point, with flow reduced by 35.79%. After these lowest flows, the proximal blood flow appeared to increase gradually without any cycling or sharp increases.

After the 10 °C treatment, proximal blood flow was at it's least between 17 and 19 min. post-immersion (a 47.8% reduction), and between 26 and 28 min. for the 5 °C immersion (a 48.05% reduction).

A significant interaction was demonstrated for the post-experimental and the experimental plus post-experimental analyses. Figure 3 illustrates these interactions. During the cooling phase, the two blood flow curves tend to diverge, with the 10 °C treatment providing a more effective reduction in blood flow. Following the removal of the cold water, the flow curves tend to converge and cross.

B. Forearm Temperature

The forearm cooling and rewarming curves depicted in figure 6, are essentially the same in profile for both the 5 °C and 10 °C treatments. They do however, differ in magnitude, as expected, with the colder water bath resulting in a lower final temperature during

immersion, and a slower rewarming curve.

Unlike Lewis' (1930), skin temperature curves for the cooling index finger, no fluctuations in temperature, or "hunting reactions" were observed for either of the experimental temperatures. As the forearm skin is not rich with arterio-venous anastamoses (Roddie, 1983), oscillations in forearm temperature were not expected. Knight et al (1980), monitored ankle temperatures during 40 min. of 1.4 °C water immersion. These researchers, observed a sudden decrease in skin temperature in the first 5 min. of cooling, followed by a gradual decrease throughout the remainder of the cooling period. The cooling and rewarming curves for the ankle, plotted by Knight et al, are remarkably similiar to the mean curves obtained from the present study.

The apparent discrepancies between distal and proximal blood flow (figures 2 and 3), and forearm skin temperature (figure 6), are noteworthy. While both proximal and distal blood flows appear to increase slightly after 10-12 min. of immersion in both experimental temperatures, forearm temperature continues to decrease throughout immersion, despite underlying blood flow changes. During the post immersion phase, the forearm temperature indicates a gradual increase in blood flow, but the flow curves indicate that blood flow remains depressed for the monitoring period. These observations tend to highlight some of the problems associated with inferring blood flow changes from temperature fluctuations.

VI. SUMMARY AND CONCLUSIONS

Purpose

As cold therapy is one of the most commonly employed treatments for athletic injuries, it is of interest to the therapist or practitioner, to know the most effective methods of treatment. The rationale for ice therapy has been described, and illustrates many of the theoretical inadequacies associated with such treatments. In particular the relative effects of cooling on cutaneous blood flow and deeper skeletal muscle blood flow, has not been documented satisfactorily. In light of the observations by Clarke, Hellon and Lind (1958), that cutaneous and skeletal muscle blood flow, respond independently to various stimuli, it was the purpose of this study, to reexamine the partitioning of forearm blood flow in response to local cooling.

Methods and Procedures

Mercury strain gauge plethysmography was employed, to monitor the effects of two local cooling treatments, on the forearm skeletal muscle, and cutaneous blood flows, of 20 male subjects. Due to attrition and incomplete data, only 16 subjects were presented for statistical analysis. The subject's right forearm was immersed in cold water on two separate occaisions, with proximal and distal forearm blood flow being monitored for the 30 min. of cold immersion (experimental), and the 30 min. following immersion (post-experimental). The treatment temperatures of 5 °C and 10 °C were presented randomly, with both treatments being experienced by all subjects.

Data Analysis

Because of intra and inter individual variability in resting blood flow values, all data was transformed into relative blood flow deviations, by subtracting the individual's mean resting blood flow from all of the experimental and post-experimental blood flow measurements. These deviations were averaged for every three minutes of the experiment. A series of two way analysis of variance tests with repeated measures over the two

treatment temperatures and 20 time segments, were used to examine the effects of treatment temperature and treatment duration on the blood flow proximally and distally.

Results

The only significant temperature effect noted, was for the proximal blood flows, during the cold immersion. During this time, muscle blood flow in the 10 °C treated forearm, was significantly less than that observed for the 5 °C treated forearm. Both treatment temperatures significantly reduced the proximal and distal blood flows, during the post-experimental period, and when the experimental and post-experimental values were analysed together as a single treatment period. No significant time effect was demonstrated for either the proximal or distal blood flows, during the cooling period. The lowest distal blood flows recorded during this experimental period were a 47.4% reduction from the mean resting flow, after 7 min. of 5 °C cooling, and a 50.39% reduction after 16 min. of 10 °C cooling. A 22.14% reduction in proximal blood flow was demonstrated after 10 min. of 5 °C cooling, and a 35.79% reduction after 13 min. of 10 °C cooling.

A reactive hyperemia in both the distal and the proximal blood flows, was observed immediately following the 5 °C cold treatment, persisting for 5-7 min. This effect was not however observed for the 10 °C treated forearm.

Discussion

Distal forearm blood flow is reduced for both temperature treatments, presumably because of the direct effects of cold on the smooth muscle of the cutaneous vasculature, and by reflex arcs, originating with the cold receptors and sensory nerves. It is postulated by the present investigator, that the 5 °C cooling was so severe at the subcutaneous level, that "chemical vasodilators" were formed, and intitiated a reactive hyperemia, when cooling ceased. Other than this reactive hyperemia, the cutaneous circulatory effects were essentially the same for the 5 °C and 10 °C treatments.

Muscle blood flow underwent a significantly greater decrease during the 10 °C treatment, than during the 5 °C treatment. It is suggested that this difference may be attributable to an axon reflex, elicited by the 5 °C treatment, which mediates a decreased vasoconstriction in the muscle circulation.

Conclusions

On the basis of the present study, the following conclusions were made.

1. The 10 °C treatment significantly reduced proximal blood flow more than the 5 °C treatment. This finding does not support research hypothesis 1.

2. Distal blood flow during the 5 °C treatment was not significantly different from the distal blood flow during the 10 °C treatment. This finding supports research hypothesis 2.

3. The proximal blood flow was reduced significantly by the 10 °C treatment and the 5 °C treatment, with time. This finding does not support research hypothesis 3.

4. The distal blood flow during the 5 °C and the 10 °C treatment was significantly reduced with time. This finding does not support research hypothesis 4.

Clinical Implications

The results of the present study indicate that the most effective reduction in cutaneous blood flow is realised after approximately 15 min. of cooling. A 5 °C coolant

does not significantly reduce cutaneous blood flow more than a 10 °C treatment, but does cause a reactive hyperemia. This reaction is probably not a desirable effect when treating an acute injury, and could be avoided by using the 10 °C treatment.

The most effective reduction in skeletal muscle blood flow, was acheived with approximately 12 min. of 10 °C immersion.

A significant reduction in both proximal and distal blood flow was observed for at least 30 min. following the 30 min. immersion in 5 °C and 10 °C water. This finding is contrary to the commonly held theory, that an "after effect vasodilation" on cold source removal, will increase local blood supply. It is suggested that overall, the 10 °C cold treatment is more effective in reducing cutaneous and skeletal muscle blood flow, without eliciting any reactive increases in flow.

Further research is required in this area to examine the effects of additional treatment temperatures, cold treatment modalities, and to manipulate treatment times, with a view to establishing an optimal cold treatment temperature, which will produce the desired results in the shortest period of time, and with minimal patient discomfort.

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APPENDIX A

Strain gauge calibrations

1. The Offner Dynograph recorder was switched on at least 30 min. prior to any calibrations or blood flow measurements.

2. With the Offner Amplifier gain set at 50 μV, and the strain gauge coupler potentiometer set at mid range (i.e. 500), a 5 Ohm resistance was introduced into the bridge via a calibration switch. The coupler was calibrated by adjusting the balance until the recording stylus was zeroed. At this point the bridge was calibrated so that a potentiometer reading of 500 = 5 Ohms .

3. The mercury in silastic strain gauge was attached to a vertical stand via the perspex mounts. The strain gauge was connected to the strain gauge coupler, and a 20 g. weight was suspended from the end of the gauge. The coupler potentiometer was adjusted, to zero the dynograph stylus, and the potentiometer reading was recorded as the resistance for that gauge under a 20 g. tension. (i.e. a potentiometer reading of 602 = 6.02 Ohms)

4. With the 20 g. weight removed, the stand was placed in a horizontal position, and the free end of the gauge was attached to a linear micrometer. The linear micrometer was initially adjusted to reproduce the same resistance recorded for the mercury strain gauge under 20 g. tension.

5. The micrometer was adjusted to produce 1.0 mm increments in gauge length, resulting in stylus deviations. From these recordings, the average pen deviation for a 1.0 mm change in the strain gauge length was calculated and recorded.

6. This procedure was repeated for each of the 6 strain gauges, with the gauges connected to the proximal and then the distal strain gauge couplers. This procedure was repeated three times throughout the study, with the average results, and strain

gauge characteristics presented in Table 12.

Table 12

Mercury Strain Gauge Characteristics

		DISTAL CHANNEL 50 μV		PROXIMAL CHANNEL 50 μV	
Gauge	Length (cm.)	Resistance for 20 g. tension	Devn. for 1 mm. stretch	Resistance for 20 g. tension	Devn. for 1 mm. stretch
A	45.0	5.75 Ohm	130.28	4.38 Ohm	142.89
B	47.5	5.76 Ohm	121.30	4.38 Ohm	132.09
C	50.0	5.69 Ohm	114.25	4.32 Ohm	128.73
D	57.5	6.34 Ohm	116.89	4.99 Ohm	131.84
E	60.0	6.87 Ohm	125.22	5.48 Ohm	136.64
F	62.5	7.06 Ohm	122.74	5.68 Ohm	136.77

APPENDIX B

Bloodflow calculation

1. The strain gauge mount was attached to the forearm in the appropriate position, and the tension was altered with the adjusting nut, to reproduce a 20 g tension.

2. Plethsymographic inflow traces were made as per the experimental procedure.

3. Ignoring the initial cuff inflation artefact, a tangent was fitted to the plethsymographic inflow trace, and the slope of this tangent Z, was calculated as mm deviation/sec.

4. This figure Z was multiplied by 60 to give Y mm deviation/min.

5. Y (mm deviation/min.) was divided by a calibration factor for the strain gauge and the recording channel used, from Table 1. This equation yielded W, the mm of strain gauge stretch/min.

6. The mm of strain gauge stretch, W, was divided by the resting circumference of the recording site on the forearm (mm), and multiplied by 100 to give V, the percentage change in circumference.

7. By multiplying the percentage circumference change by a factor of two (2), the percentage change in volume is given. This percentage of volume change, equals the forearm blood flow at that point, and is given in terms of ml/100 ml of tissue/min.

APPENDIX C

Temperature effect on mercury strain gauge sensitivity

To ascertain the possible effects of the experimental temperatures on the mercury strain gauge sensitivity, two trials were carried out to monitor changes in the strain gauge function.

For these trials, a water filled bladder was suspended in the perspex water bath in the position that the forearm would normally occupy. The rubber bladder was filled with room temperature water, and was connected to a 2 ml. pyrex syringe. Strain gauge "A" was assembled in the perspex strain gauge mount, and was applied to the bladder in the same manner as application to the forearms of subjects. The gauge was connected to the dynograph recorder, and the tension was adjusted to 20 g. The syringe was used to increase the bladder volume by 1 ml, giving a corresponding deflection of the recording stylus. One ml of volume was then withdrawn from the bladder. This lengthening and shortening of the gauge was repeated 5 times for each condition.

The procedure was carried out initially with the water bath empty, and then experimental temperature water was introduced into the bath for 30 min. as per the experimental procedure. After 30 min. the water was drained from the water bath and volume changes were made for a further 30 min. Volume deviations were made every 5 minutes for the entire 60 min.

The percentage difference between the deviation caused by a 1 ml volume change was calculated at room temperature and for each of the experimental time segments were calculated, and were used as measures of the temperature effect on strain gauge sensitivity. The results from these trials are contained in Table 13.

The percentage error in estimation of changes in circumference caused by temperature effects on the sensitivity of the strain gauges, ranged from 0.03% to 2.81%. These errors of estimation were all overestimations for the 10 °C trials, and underestimations for the 5 °C trials. This suggests that the differences were as much due to experimental error, as they were to a distinct temperature effect.

The net effect of these effects is a potential error of .06% to 5.62% in blood flow estimation. No direction can be assigned to this error, but it is not considered significant, in light of the magnitude of changes in blood flow observed during cooling.

Table 13

Temperature effect on strain gauge sensitivity

	5 °C IMMERSION		10 °C IMMERSION	
TIME	MEAN DEVN.	% DIFFERENCE	MEAN DEVN.	% DIFFERENCE
Rest	40.54		36.04	
		Cold water added		
5 min.	-	-	35.58 mm	1.28
10 min.	40.44 mm	.25	36.67 mm	1.75
15 min.	39.85 mm	1.7	36.50 mm	1.28
20 min.	39.95 mm	1.46	36.00 mm	.11
25 min.	40.38 mm	.39	35.70 mm	.94
30 min.	40.05 mm	1.21	36.33 mm	.80
		Cold water removed		
35 min.	39.96 mm	1.43	36.08 mm	.11
40 min.	39.54 mm	2.47	36.14 mm	.28
45 min.	39.82 mm	1.78	36.05 mm	.03
50 min.	39.40 mm	2.81	35.68 mm	1.00
55 min.	40.50 mm	.10	35.72 mm	.89
60 min.	39.67 mm	2.17	35.73 mm	.86

APPENDIX D

Informed consent form

Informed consent form for investigative study:

"FOREARM BLOOD FLOW WITH LOCAL COLD APPLICATION"

SUBJECT CONSENT

I_____ do hereby agree to participate in the study entitled "Forearm blood flow with local cold application" conducted by Phil. Handcock.

I do not/have not suffer(ed) from peripheral vascular problems, blood pressure abnormalities or cardiovascular anomalies.

The investigator has cautioned me, as to the potential risks of the study, and of the sensations that I can expect to experience during cold water immersion. I have been advised that I may withdraw from the study at any time.

_____ _____

SUBJECT'S SIGNATURE DATE

I was witness to the above explanation and signature

_____ _____

WITNESS' SIGNATURE DATE